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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Crops Research Division serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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(3) ABSTRACTS are requested for all except very short articles.

(4) CAUSES OF DISEASES should be named. For bacteria, fungi, nematodes, etc., give the Latin name of the organism; for viruses either or both the accepted common name of the virus or a Latin name if you prefer it and there is one; for non-parasitic diseases state the causal factor if it is known. If the cause of a disease has not been determined say so.

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ACCEPTANCE OF MANUSCRIPTS

The increase in the volume of pertinent material offered for publication in the Plant Disease Reporter has made it necessary to limit the subject matter and the length of articles accepted. The subject matter should emphasize new things in plant pathology, such as new records of disease occurrence, serious outbreaks and epidemics, conditions affecting development of plant diseases, techniques of investigation including instrumentation, new discoveries in control including new materials and their evaluation. Manuscripts will be limited to 15 double-spaced typed pages. Insofar as possible, material should be presented as graphs rather than tables. Papers cannot be accepted for publication that report routine control experiments, reviews, bibliographies without annotation, results of routine surveys, mere summaries or lists of plant diseases. By following this procedure we hope to continue publishing all articles promptly.

Paul R. Miller

Manuscripts for and correspondence about this publication should be sent to:

PLANT DISEASE REPORTER
Mycology and Plant Disease Reporting Section
Crops Protection Research Branch
Plant Industry Station
Beltsville, Maryland

THE PLANT DISEASE REPORTER

Crops Research Division — Plant Industry Station, Beltsville, Maryland
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New or Unusual Records of Plant Diseases, page 1301: Tobacco diseases in Panama, by G. B. LUCAS. Stinking smut in Virginia, by S. B. FENNE.

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PREVENTION OF ANTIBIOTIC INJURY WITH Na-K-CHLOROPHYLLIN

Peter A. Ark[1] and James P. Thompson[2]

Abstract

Injury caused by Acti-dione, aureomycin, patulin, streptomycin, or Terramycin can be fully prevented by adding one percent of Na-K-chlorophyllin to the antibiotic before its use on the plants.

INTRODUCTION

Dekker and Ark (1) reported that Na-K-chlorophyllin protected the antibiotics, pimaricin and GS1, from the destructive action of ultraviolet rays. The injury produced by antibiotics is of importance to plant pathologists since many known antibiotics, while possessing remarkable microcidal properties, are deplorably phytotoxic. Therefore, agents which circumvent this phenomenon may enhance practical value of such antibiotic preparations. This is a brief report on prevention of injury caused by various antibiotics by combining them with Na-K-chlorophyllin. Wilson and Ark (2) found that when Pinto beans were sprayed with 10 ppm Acti-dione the plants showed signs of injury which were absent when Na-K-chlorophyllin was added to the Acti-dione spray.

MATERIALS AND METHODS

Aqueous solutions of the following antibiotics were used: Acti-dione, aureomycin, patulin, streptomycin sulfate and Terramycin. In one series of experiments, greenhouse-grown Pinto beans and Marketer cucumbers sprayed with appropriate concentrations of the antibiotics were compared with similar plants sprayed with the same concentrations of the antibiotics plus 1 percent Na-K-chlorophyllin. The plants were set on a bench in strong light since it was known that the symptoms of injury from antibiotics are most clearly expressed under that condition.

RESULTS AND CONCLUSIONS

Both the beans and the cucumbers sprayed with Acti-dione, 25 ppm, showed a pronounced mottling and mosaic type of injury after 3 days. A necrotic type of injury was observed on plants sprayed with patulin, 100 ppm, and a chlorosis of varying degrees of severity was present on plants that received aureomycin, 500 ppm, streptomycin, 1000 ppm, and Terramycin, 500 ppm. This sensitiveness to the antibiotics was not present in all plants sprayed with the same concentrations of the antibiotics to which 1 percent of the Na-K-chlorophyllin was added. A 0.2 percent Na-K-chlorophyllin concentration gave only partial protection from the injury.

It appears that all experimental plants were sensitive to the high levels of the antibiotics and in all cases full protection from injury was obtained with the concentration of the Na-K-chlorophyllin used. A lower concentration of the chlorophyllin is not effective in eliminating the injury.

Since Na-K-chlorophyllin is not a pure substance, it can be assumed that the anti-injury factor in the chlorophyllin is in a rather low concentration. The anti-injury factor does not appear to be specific, as it affords equal protection in cases of both necrotic and chlorotic types of injury (Figs. 1, 2, and 3).

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[1] Professor of Plant Pathology, University of California, Berkeley 4, California.

[2] Laboratory Technician II, University of California, Berkeley 4, California.

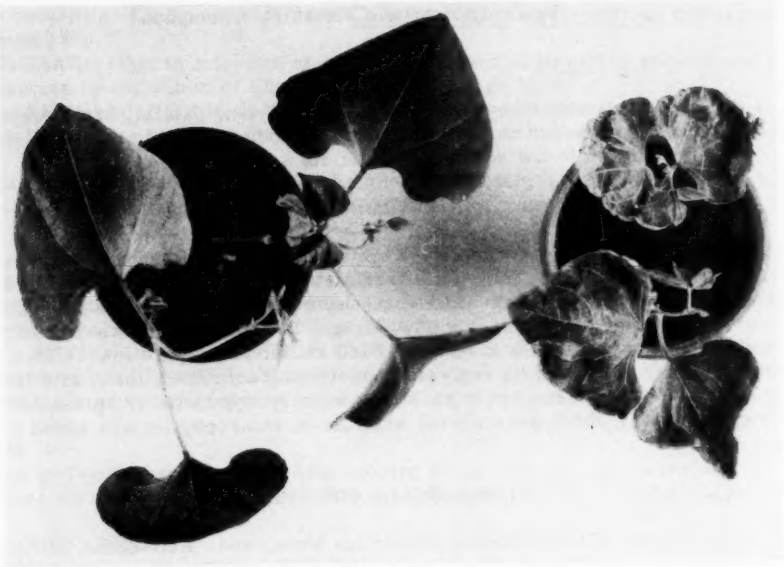
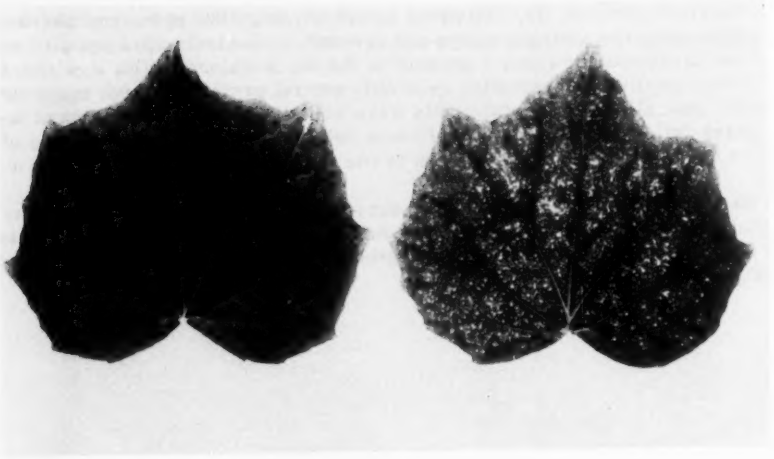


FIGURE 1. Injury on Pinto bean leaves caused by Acti-dione, 25 ppm spray -- right; prevention of injury by adding to the Acti-dione spray, Na-K-chlorophyllin, 1000 ppm -- left.



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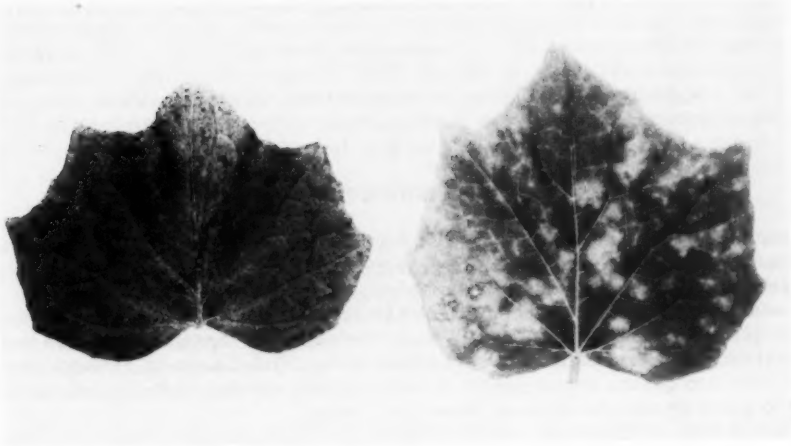

FIGURE 3. Injury on cucumber (Marketer) leaves caused by spraying with streptomycin, 1500 ppm -- right; prevention of the injury by adding to the streptomycin spray Na-K-chlorophyllin, 1000 ppm -- left.

Literature Cited

1. DEKKER, J., and PETER A. ARK. 1958. Prevention of oxidative destruction of the antifungal antibiotic, pimaricin, by chlorophyll, ascorbic acid, and other compounds. Phytopathology 48. (Abst.) In press.
2. WILSON, EUGENE M., and PETER A. ARK. 1958. The effect of chlorophyll on the phytotoxicity of Acti-dione on bean. Plant Disease Reptr. 42: 1069-1070.

DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA, BERKELEY 4, CALIFORNIA

COMPARATIVE ANTIBIOTIC ACTIVITY OF THREE BRANDS OF AGRICULTURAL STREPTOMYCIN

Peter A. Ark[1] and Donald C. Hildebrand[2]

Abstract

Agricultural streptomycin derived from the three brands made in this country did not show any significant differences when applied to the plants either as wettable or dust formulations.

INTRODUCTION

In recent years streptomycin has received a great deal of attention from growers of the various crops that are subject to attacks by plant pathogens that are controllable by it. The main source of agricultural streptomycin available to farmers is supplied by Chas. Pfizer & Co., Inc. under the names Agri-mycin 100 and Agri-mycin 500; Merck, Sharp, and Dohme -- Agristrep; and Mathieson and Co. -- Phytomycin. Because each brand is compounded in different types of extenders, experiments were performed to establish comparative value of these brands of streptomycin into quality performance of both wettable and dust formulations made from them. This paper reports on results obtained from these tests.

MATERIALS AND METHODS

The streptomycin brands used in the experiments were: Agri-mycin 100, Agri-mycin 500, Agristrep (47 percent and 54 percent of streptomycin) and Phytomycin. To make wettable formulations, the material was dissolved in distilled water. To make dust formulations, the material was blended with a Nuclay carrier (Kennedy Minerals Co., Inc., Los Angeles, California) and passed three times through a Wiley mill with a 60 mesh screen. The test plants were greenhouse-grown cucumbers (Marketer var.) in a 3- or 4-leaf stage. In dusting experiments the plants were covered with fine mist before the dusts were applied. All plants in wettable and dust series were left on the greenhouse bench for 24 hours after the treatment; after which, the leaves were detached for bioassaying purposes. The detached leaves were divided into two groups. In one group the leaves were not washed. In the second group the leaves were washed thoroughly with tap water, while brushing gently with a very soft camel hair brush (2 inches wide), and then were placed in the running water for 10 minutes. After the washing excess moisture was absorbed with Kleenex tissue and the leaves were frozen in a freezer, usually overnight. Next day the leaves were thawed and the juice extracted with a plant juice press, supplied by G. B. Salm and P. J. Kipp and Zonen, Keisersgracht 642-44, Amsterdam, Netherlands. Filter paper bioassaying discs, SS-740E, were dipped in the plant juice in such a way that the discs were saturated evenly and the excess of the juice was removed by touching the edge of the disc on the inside wall of the beaker. The prepared discs were placed on potato-dextrose-peptone agar plates, seeded previously with an 18-hour-old culture of Erwinia amylovora, and dried for 20 minutes at 40°C. The plates with the discs were incubated for 24 hours at 28°C, and inhibition zones were expressed in millimeters of the diameters. The brands of streptomycin used in the experiments were compared also as to size of inhibition zones, with a solution (made from each of the brands) containing 100 ppm of the same brand of streptomycin.

RESULTS

Inhibition zones on bioassay plates from discs saturated with the solutions containing 100 ppm streptomycin were as follows (mm - dia.): Agri-mycin 100 -- 31.1; Agri-mycin 500 -- 28.8; Agristrep (47 percent) -- 29.8; Agristrep (54 percent) -- 30.1; and Phytomycin -- 29.7. Bioassaying of the juice of washed cucumber leaves sprayed with a 100 ppm solution of streptomycin showed the following inhibition zones (mm -- dia.): Agri-mycin 100 -- 23.5; Agri-mycin 500 -- 21.5; Agristrep (47 percent) -- 21.4; Agristrep (54 percent) -- 22.4; and Phytomycin -- 23.9. For the unwashed leaves in the sprayed series the inhibition zones were

[1] Professor of Plant Pathology, University of California, Berkeley 4, California.

[2] Research Assistant, Department of Plant Pathology, University of California, Berkeley 4, California.

somewhat larger as follows (mm -- dia.): Agri-mycin 100 -- 31.0; Agri-mycin 500 -- 30.2; Agristrep (47 percent) -- 32.3; Agristrep (54 percent) -- 31.6; and Phytomycin -- 29.1. In the dust series the results for the washed leaves were as follows (mm -- dia.): Agri-mycin 100 -- 24.5; Agri-mycin 500 -- 22.6; Agristrep (47 percent) -- 27.5; Agristrep (54 percent) -- 26.2; and Phytomycin -- 22.6. For the juice from the unwashed leaves: Agri-mycin 100 -- 33.1; Agri-mycin 500 -- 33.8; Agristrep (47 percent) -- 36.2; Agristrep (54 percent) -- 33.1; and Phytomycin -- 34.7. The zones of inhibition for the solutions containing 100 ppm of streptomycin prepared from the dust formulations were as follows: Agri-mycin 100 -- 25.9; Agri-mycin 500 -- 25.8; Agristrep (47 percent) -- 28.7; Agristrep (54 percent) -- 26.4; and Phytomycin -- 25.3.

CONCLUSIONS

It appears that the agricultural grades of streptomycin as represented by the three major brands do not differ significantly in their behavior when tested from aqueous solutions containing 100 ppm or from the juice extracted from cucumber leaves treated either with wettable or dust formulations. The variation in size of inhibition zones appearing in the experimental data can be due to many factors, such as variation in the uniformity of the product, the effect of a carrier employed in standardizing a given brand of streptomycin, or the age of the antibiotic used in the experiments.

DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA, BERKELEY 4, CALIFORNIA

PHYTOACTIN SHOWS ACTIVITY AGAINST PODOSPHAERA LEUCOTRICHA[1]

Roderick Sprague[2]

In preliminary trials small samples of a new antibiotic, Phytoactin, and a companion material, Phytostreptin[3], were furnished by Dr. Jack Ziffer of the Pabst Laboratories.

Because we had a number of small potted pear seedlings available, and very heavily mildewed, trials were run in February 1958. The concentrated material was dissolved in methanol and diluted with distilled water with a trace of B-1956 as a wetter and sprayed with a "trombone" type of tube hand-sprayer. Counts were taken before spraying on February 13th, after spraying on February 14th, and again on February 25th. Most of the mildew was dead after spraying. However, on teriminals that were coated with mildew, some recovery had occurred on February 25th. A second spray on February 25th killed all of the remaining mildew in the Phytoactin trial, but in one plant sprayed with Phytostreptin mildew was returning on young leaves. New growth in the trees sprayed with Phytoactin were still healthy on March 5th. The results of this preliminary trial are shown in Table 1.

Table 1. Results of spraying small potted seedling pears infected with powdery mildew in the greenhouse at the Tree Fruit Experiment Station, Wenatchee, Washington, on February 13, 1958, and again on February 25, 1958.

Material	Rate	Number of leaves per tree							
		February 13		February 14		February 25		March 5	
		Diseased	Healthy	Diseased	Healthy	Diseased	Healthy	Diseased	Healthy
Phytoactin	100 ppm	4	0	0	4	0	4	0	4
		6	2	0	8	0	8	0	8
		20	1	1	20	12	16	0	21
		14	6	0	20	1	19	0	15
	Total	44	9	1	52	13	47	0	48
Phytostreptin	100 ppm	23	0	0	23	0	24	0	24
		2	0	0	2	0	2	0	2
		26	0	0	26	2	24	0	36
		16	0	2	14	6	10	3	14
		9	0	0	9	0	9	0	9
	Total	76	0	2	74	8	69	3	85
Unsprayed		19	3	19	3	19	4	19	4
		11	1	11	1	11	1	10	2
		10	4	10	4	10	4	9	5
		11	3	11	3	11	3	11	3
		4	3	4	3	4	3	4	3
	Total	55	14	55	14	55	15	53	17

Because the first results were promising, a liquid concentrate of Phytoactin (L-307) of a less expensive and cruder form was used on the greenhouse pears. The action of the new batch at 100 ppm was slower than with the earlier purified product, but was still promising. Actual loss of leaf surface by mildew was 88.9 percent for eight seedlings before spraying on June 13th. On June 26th, after spraying on the 13th, the loss was down to 21.3 percent. The seedlings were again sprayed with 100 ppm on the 26th. On July 17th the loss of surface was down to 8 percent. Since this showed considerable promise, the Pabst Laboratories furnished us with a larger quantity of the cruder concentrate. We applied this on 8-year-old Black Jona-

[1]Washington Agricultural Experiment Stations, Pullman, Washington. Contribution from Department of Plant Pathology. Project No. 1164.

[2]Plant Pathologist, WSC Tree Fruit Experiment Station, Wenatchee, Washington.

[3]Ziffer, Jack, S. J. Ishihara, T. J. Cairney, and A. W. Chow. 1957. Phytoactin and Phytostreptin, two new antibiotics for plant disease control. Phytopathology 47: 539 (Abst.).

thans in our mildew block on June 30th, using 200 ppm. Spray was applied by hand-gun through a stationary spray outfit. Irrigation water was used. This water has a pH of 6.8 and is faintly turbid with soil. The results are shown in Table 2.

Table 2. Results of spraying Black Jonathan with Phytoactin (200 ppm) June 30, 1958, in the mildew plots at Tree Fruit Experiment Station, Wenatchee, Washington.

Tree no.	Before spraying June 30			After spraying July 7			July 22		
	Percent mildew	Factor	Calculated loss	Percent mildew	Factor	Calculated loss	Percent mildew	Factor	Calculated loss
9-2	46.7	3	15.6	22.4	4	5.6	4.4	4	1.1
9-4	22.3	3	7.4	12.2	4	3.1	9.4	4	2.4
9-6	24.4	3	6.1 [8.1]	20.2	4	5.1	8.5	4	2.1
9-8	43.5	2 1/2	17.4	18.5	4	4.6	14.2	4	3.8
Av.	34.2	2.9	11.8	18.4	4	4.6	9.1	4	2.4

The factors (which range from 1=severe, to 5=light) shown in Table 2 are based on estimated leaf surface covered by mildew. Calculated loss is approximated by dividing the percent of mildewed leaves by the factor. The antibiotic also proved very promising in the field. Many of the mildewed terminals had dried and were not reinfected. A few terminals had started to send out new leaves; others had reinfected lightly. The fact that the second count was taken 22 days after spraying indicates that the material held up well. Phytoactin warrants further trials.

WASHINGTON AGRICULTURAL EXPERIMENT STATIONS, PULLMAN, WASHINGTON

CROWN GALL CHEMOTHERAPY WITH TERRAMYCIN[1]

Ira W. Deep

Summary

Incipient crown gall infections were eliminated from 1-year-old Mazzard cherry trees by a 400 ppm treatment with Terramycin for 30 minutes. An 800 ppm treatment with Terramycin for 15 minutes effectively prevented infection by crown gall bacteria when the trees were planted in infested soil. Neither of these treatments were phytotoxic in the instance cited, but they have been damaging in other trials. The preplanting treatment now recommended in Oregon for control of crown gall on Mazzard cherry is a 30 minute soak in 200 ppm of Terramycin.

Crown gall, caused by Agrobacterium tumefaciens (E. F. Smith and Towns.) Conn, is particularly troublesome in the nursery where propagation wounds serve as sites of penetration and infection. Treatment of propagation wounds with chemicals to prevent infection has been a standard procedure, but the mercurial compounds used have been phytotoxic to some plants. During an earlier investigation (3) three antibiotic formulations, Terramycin, Agrimycin, and streptomycin sulfate, were tested and compared with Semesan Bel as disinfestants of wounds on Mazzard cherry, (Prunus avium L.). Terramycin was much more effective than the other materials tested.

The crown gall problem on Mazzard cherry is frequently complicated by two factors. Many of the seedling trees have incipient infections but no detectable galls when they are transplanted. Complete surface disinfestation prevents infection during root-pruning procedures but does not eliminate incipient galls. A second factor is that much of the nursery soil is infested with crown gall bacterium. Surface disinfestation of wound sites loses much of its value when the trees are immediately planted in heavily infested soil. The ideal treatment would thoroughly disinfest roots, eliminate galls, and provide sufficient residual effect to prevent infection by bacteria in the soil.

Terramycin treatments are potentially more beneficial than strictly surface-acting treatments because there is a possibility of chemotherapeutic activity against both the causal bacterium within host tissues and the developing galls. Klemmer, Riker, and Allen (5) reported the chemotherapeutic action of Terramycin in completely inhibiting gall formation on tomato and marigold. The antibiotic was administered by immersing roots of the test plants in aqueous solutions of the antibiotic. Following repotting the plants were inoculated by adding 0.005 ml of bacterial culture to punctures made through each of the four internodes along the main stem. Activity of the antibiotic consisted apparently in suppressing development of gall tissue rather than in killing the bacterium. Janke and Granits (4) reported breakdown of geranium crown gall tissue following application of Terramycin to the surface of galls.

This paper reports results from greenhouse experiments designed to determine whether Terramycin could act chemotherapeutically by eliminating incipient infections from Mazzard cherry trees and whether Terramycin treatments would prevent infection of the trees by bacteria present in the soil.

PROCEDURES AND RESULTS

Elimination of Incipient Infections

One hundred and sixty Mazzard seedling trees were root-pruned with shears that were dipped in a 25 percent Clorox solution (5.25 percent sodium hypochlorite) prior to pruning each tree. The roots were washed in soapy water, surface sterilized by dipping in 20 percent Clorox for 5 minutes and allowed to dry. Eighty of the trees were planted in sterilized soil in number 10 cans and 80 were treated for 30 minutes in 400 ppm of Terramycin[2] prior to

[1] Technical Paper No. 1182, Oregon Agricultural Experiment Station.

[2] The hydrochloride formulation of Terramycin containing 83.5 percent active ingredient was used in all trials. Terramycin was supplied by Chas. Pfizer & Co., Inc.

planting. The trees were grown in the greenhouse for 5 months, then dug and examined for galls.

Sixty-seven of 80 trees survived in both the treated and untreated blocks. Fifteen of the untreated trees were infected with crown gall while none of the trees which had been treated with 400 ppm of Terramycin for 30 minutes were infected (Table 1). Since other sources of infection were removed by complete surface disinfestation and sterilization of all soil used, the data indicate chemotherapeutic activity in eliminating incipient infections. These results support data obtained in a previous trial with Agri-mycin (3).

Table 1. Effectiveness of Terramycin in eliminating incipient crown gall infections on Mazzard cherry.

Treatment	Row	Number of plants: With gall	Number of plants: Without gall	Percentage gall
Trees root pruned, surface sterilized and planted in sterilized soil. (The uninoculated, untreated control)	1	5	12	29.4
	2	2	11	15.4
	3	3	15	16.8
	4	5	14	26.3
Totals		15	52	22.4
Trees root pruned, surface sterilized, treated with 400 ppm of Terramycin for 30 minutes, and planted in sterilized soil	1	0	18	0
	2	0	16	0
	3	0	17	0
	4	0	16	0
Totals		0	67	0

Preventing Infection of Transplanting Stock by Bacteria in the Soil

One hundred and sixty Mazzard seedling trees were root pruned and treated with Terramycin, 80 at 400 ppm for 30 minutes and 80 at 800 ppm for 15 minutes. These trees, along with 80 which were root pruned but given no treatment, were planted in number 10 cans using soil which had been artificially infested with a culture of Agrobacterium tumefaciens previously isolated from Mazzard cherry. After 5 months the plants were examined for galls.

Forty-five of the 80 trees which had been treated with 400 ppm of Terramycin for 30 minutes survived. This compared to 63 following an 800 ppm Terramycin treatment for 15 minutes and 60 in the control. Of the 45 survivors in the 400 ppm treatment five had crown gall while only one of the 63 survivors of the 800 ppm treatment had gall. Thirty-one of the 60 control plants were infected (Table 2). The 800 ppm treatment was extremely effective in preventing subsequent infection by bacteria in the soil, and there was no phytotoxicity. The 400 ppm treatment was less effective and was phytotoxic. This same treatment, however, was used in the experiment summarized in Table 1 with no indication of phytotoxicity.

DISCUSSION

Root-dip treatments with Terramycin hydrochloride were effective in eliminating incipient crown gall infections and in preventing infection when trees were planted in infested soil. The antibiotic may have prevented gall development in two ways, 1) by suppressing development or causing breakdown of incipient galls, or 2) by killing bacteria present in the roots. It has been shown that killing the bacterium after establishment of infection does not prevent development of a gall (6), and it is unlikely that in every instance in which chemotherapeutic activity occurred the bacterium was present but galls had not been initiated. Klemmer, Riker and

Table 2. Effectiveness of preplanting treatments of Mazzard cherry trees in preventing infection from artificially infested soil.

Treatment	Row	Number of plants: With gall	Number of plants: Without gall	Percentage gall
Terramycin hydrochloride 400 ppm for 30 minutes	1	1	11	
	2	1	13	
	3	0	9	
	4	3	7	
Totals		5	40	11.1
Terramycin hydrochloride 800 ppm for 15 minutes	1	0	16	
	2	0	13	
	3	1	16	
	4	0	18	
Totals		1	63	1.6
Control	1	8	7	
	2	4	12	
	3	8	6	
	4	11	4	
Totals		31	29	51.7

Allen (5) reported a suppressed development of gall tissue following inoculation of the stem and treatment of roots of tomato and marigold plants with Terramycin.

Prevention of infection following treatment and planting in infested soil may have been the result of the antibiotic being taken into the tissues or the residue of antibiotic at the surface of the roots. In either case the trees must have been protected for several days prior to the formation of callus tissue. Braun (2) reports that infection of periwinkle occurs only during the 4-day period following wounding. Preliminary work suggests that the susceptible period might be considerably longer for certain plants since 1-year-old Mazzard cherry trees became infected when inoculated 8 days after root pruning.

The chemotherapeutic activity of Terramycin and related tetracycline compounds against crown gall has been reported previously, but this is the first report of such activity on an economically important host using a treatment which could be applied by the grower. In earlier trials (3) Terramycin was shown to be effective in surface disinfestation of roots. The occurrence of three types of activity by the antibiotic, chemotherapy, protection of wounded areas against bacteria in the soil, and disinfestation of roots, indicates that such treatments may significantly reduce losses due to crown gall on Mazzard cherry rootstocks.

Preliminary work with an agricultural grade of Terramycin suggests that it will be somewhat less effective but also less phytotoxic than the hydrochloride form. Further tests are being made to determine the most effective treatment that can safely be applied.

Ark and Bingham (1) have reported results with an agricultural grade of tetracycline which are very encouraging although the authors appear rather conservative at present about the benefits of their treatments. Mazzard cherry rootstocks obtained from an area where the soil was heavily infested with the crown gall bacterium were treated with 100 and 200 ppm of tetracycline for 1 hour. Control trees which received no treatment were also planted. When dug after several months 3.6 and 5.2 percent of the plants treated with 100 and 200 ppm of tetracycline, respectively, were infected while the incidence of infection in the corresponding control trees was 18.8 and 17.9 percent. It was suggested that the reduction in gall represents an elimination of incipient infections, but the trees were handled under grower conditions, and some of the 18 to 19 percent infection in the control might have resulted from bacteria present on the surface of the planting stock or in the soil, or from spread of the bacterium

from one plant to another on pruning shears. Since many of these bacteria would be on the surface of the planting stock, an antibiotic treatment could effectively eliminate them, thus reducing the percentage infection. In order to demonstrate chemotherapeutic activity by a reduction in incidence of incipient infections, other sources of infection must be removed by surface sterilization of roots and pruning shears and by planting in sterilized soil.

Regardless of the source of infection, the reduction in incidence of gall following the treatments reported by Ark and Bingham would greatly benefit the nurseryman. The data indicate a potential saving of 127 to 152 trees per thousand, and such trees are usually worth between $0.75 and $1.35 each. The cost of the chemical to treat 1000 trees would be approximately $12.00 to $15.00.

Oregon nurserymen are now advised to treat Mazzard cherry rootstocks in 200 ppm of agricultural grade Terramycin for 30 minutes. Such a treatment should be effective against the inoculum found in nature. The treatments used in this investigation, 400 ppm for 30 minutes and 800 ppm for 15 minutes, are not recommended because of the danger of phytotoxicity. These treatments have appeared safe in most trials, but phytotoxic in some. The phytotoxic level is probably dependent upon a number of factors such as size and vigor of plants, severity of root pruning, and degree of dormancy as indicated by bud-breaking.

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OREGON AGRICULTURAL EXPERIMENT STATION, CORVALLIS

RELATIVE IMPORTANCE OF CRANBERRY ROT FUNGI DURING THE STORAGE AND HARVEST SEASONS IN MASSACHUSETTS, 1956-1957[1]

Bert M. Zuckerman[2]

Abstract

The relative importance of cranberry fruit rot fungi in Massachusetts in 1956-1957 was compared with the importance of the same fungi as reported in 1916-1917 and 1926-1929. Glomerella cingulata vaccinii, which has been the most serious cranberry pathogen in the past, was also the most abundant fungus found in the current studies. Sporonema oxycocci is currently a more abundant fruit pathogen than in the past, and the relative importance of several other rot organisms has changed. Howes and Early Black, the two most important varieties in Massachusetts, did not appear to be resistant to any of the rot fungi. Though wet, humid conditions during bloom are thought to be a prerequisite for serious infection of fruit rot organisms, there did not appear to be a relationship between these weather conditions and the subsequent keeping quality of the berries. Altering of the time of bloom through "late water" and "early water" holding management practices did not significantly affect the quality of the berries. "Late water" and "early water" holding was not consistently related to the specific rot fungi found in berries at harvest. An analysis of external disease symptoms, as related to the species of fungus cultured from the berry, confirmed earlier reports that it is impossible to distinguish between cranberry rots without resorting to artificial cultures.

Studies of the relative importance of cranberry rot organisms in Massachusetts were made by Rudolph and Franklin (2) in 1916-1917, and by Shear, Stevens, and Bain (3) in 1926-1929. Since the time these studies were made, the cranberry grower has used increasing amounts of fertilizer, resulting in heavier vines and larger crops, and more water for frost protection and irrigation. Experimental evidence and field observations indicate that these practices also contribute to weaker or poorer quality fruit. Presumably this is brought about through the creation of more favorable infection and incubation conditions for the fruit rot fungi. Since cultural practices have changed markedly since the time of the previous studies, a similar study was conducted in 1956-57. This paper records the results of this work.

MATERIALS AND METHODS

Cranberries which showed evidence of decay were placed in 70 percent ethyl alcohol for several minutes and then passed through the flame of a bunsen burner. An incision was made in the skin and a portion of the pulp removed with sterile forceps was placed on a Petri plate containing potato-dextrose agar. Cultures were incubated at 23° C until they could be identified.

The data from this experiment were recorded on McBee hand-sort index cards, with the following information for each culture:

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[1] Contribution No. 1168 of the University of Massachusetts College of Agriculture Experiment Station, Amherst, Massachusetts.

[2] Associate Professor, Department of Entomology and Plant Pathology, University of Massachusetts.

1. Fungus name.
2. Berry variety from which isolate was made.
3. Bog on which cranberry was grown.
4. Macroscopic symptoms of decay.
5. Water management of bog on which cranberry was grown.
6. Date of culture.
7. Type of fungicide treatment applied in current year, if any.
8. Whether berry decayed in the field or in storage.

A section was also included which recorded the portion of the cranberry plant from which the isolation was made; however, this information is not included since this paper reports only on isolations made from berries. This paper deals exclusively with field and storage rots; consequently, only the data collected during the period of August to December are presented. The figures presented are based on 1244 determinations made during 1956-57.

The cranberries used in these studies were gathered from 39 plantings of bog. The term "field rot" refers to berries that were decayed at the time of harvest, and "storage rot" to berries that broke down after harvest. Berry samples were kept in a common storage which was arranged to approximate commercial storage conditions. It is common practice to maintain storage temperatures at above freezing, but below the average fall daylight temperatures. This is effected by the use of fans, by the practice of opening windows in storage areas at night, and, infrequently, by the insulation of storage sheds.

Fungi are listed by species names in Table 1, and are referred to thereafter by generic names.

Table 1. Occurrence of fungi in cranberries in Massachusetts during the months of August through December, 1956-1957.

Fungi	1956	1957	Total	Relative importance based on number of determinations
Acanthorhynchus vaccinii Shear	-	9	9	
Coniothyrium fuckelii Sacc.	1	-	1	
Dematium spp.[a]	-	29	29	7
Diaporthe vaccinii Shear	11	19	30	6
Diplodia vaccinii Berl. & Roum.	-	1	1	
Glomerella cingulata vaccinii Shear	90	377	467	1
Godronia cassandrae Peck	41	30	71	4
Guignardia vaccinii Shear	27	107	134	3
Helminthosporium inaequale Shear	1	-	1	
Penicillium spp.	-	3	3	
Pestalotia vaccinii (Shear) Guba	7	33	40	5
Rhabdospora oxycocci Shear	-	2	2	
Sporonema oxycocci Shear	74	167	241	2
Stemphylium ilicis Tengw.	3	-	3	
Sterile	79	119	198	
Unidentified[b]	11	3	14	
Total	345	899	1244	

[a] Dematium spp. were isolated in 1956; however, it was not until 1957 that this genus was positively recognized by the author.
[b] Records of the number of unidentified cultures were incomplete.

RESULTS

Relative Importance of Fungi

The relative importance of the six most abundant fruit rot organisms is given in Table 1. Glomerella and Sporonema made up more than 65 percent of the positive isolations. Guignardia and Godronia were of lesser importance, and Pestalotia, Dematium and Diaporthe were found infrequently. Seven additional species or genera of fungi were found but were isolated only a few times.

In 1931, Shear et al. (3) stated that Godronia was the most important of the late storage rots in Massachusetts, and Glomerella the most important of the early rots. They considered Diaporthe and Guignardia as being important early rots.

Rudolph and Franklin (2), in a study of cranberry storage rots in 1916, noted that Godronia was the most important storage rot on Early Blacks and Howes. Diaporthe and Glomerella were listed as important early rots, and Sporonema as an early rot of little significance. This study was repeated in 1917 with approximately the same results.

Figures 1 and 2 record results of the current studies as compared with those obtained by Shear et al. (3) and by Stevens and Bain (4) in the period of 1926-1929. In 1956-1957, Guignardia and Godronia were more abundant as late rots and Diaporthe as an early rot. Glomerella, the most abundant pathogen during these years, showed up most frequently as an early rot but also was a very important late rot. The current importance of Sporonema as a fruit rot pathogen is brought out by these figures. In 1956-1957, it was second only to Glomerella as a cause of cranberry spoilage. In 1916-1917 and 1926-1929, Sporonema was considered a pathogen of minor importance in Massachusetts[3].

An important difference between these data should be noted. The earlier data were based on isolations made from the Howes and McFarlin varieties of cranberries, whereas 95 percent of the current data is based on isolations made from the Howes and Early Black varieties. Over the years the McFarlin variety has decreased in importance in Massachusetts; consequently, only 3 percent of the 1956-1957 isolations were made from berries of this variety.

Determinations listed as sterile in Table 1 were isolations made from apparently rotted berries which developed no growth. Since only a portion of each berry was cultured, this does not mean that all of these berries were affected by physiological or sterile breakdown. However, these results suggest that physiological breakdown is an important factor in cranberry deterioration. Shear et al. (3) discussed sterile breakdown and considered it of considerable importance in the spoilage of cranberries.

Cranberry fungi generally vary in abundance from year to year. Whether or not this increase in the incidence of Sporonema and decline of Godronia represents a trend which can be related to changing cultural practices should be determined through continued study.

Relative Abundance of Cranberry Rot Organisms on Two Cranberry Varieties

Early Blacks and Howes are the two most important cranberry varieties grown in Massachusetts, currently accounting for 95 percent of the acreage in production. Rudolph and Franklin (2), in 1916-1917, made the following observations:

1. Sporonema was found chiefly on Early Blacks.
2. Diaporthe was more common on Early Blacks.
3. Glomerella occurred more often on Howes.
4. Godronia was prevalent on both Early Blacks and Howes as a storage rot during both of these years.

The study by Shear et al. (3), which covered the years 1926 through 1929, included Howes but not Early Blacks. This paper will concern itself only with their findings relative to Howes. Glomerella was an important early rot on Howes, and Godronia an important late rot. Guignardia was also of significance as an early rot on Howes. Sporonema was of little importance on this variety during these years.

It is of interest to compare the current findings with those of the past. These figures are

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3 Figure 1 is a reproduction of Fig. 39, pg. 35, U. S. D. A. Tech. Bul. 258. It has been copied for the purpose of comparison with Figure 2.

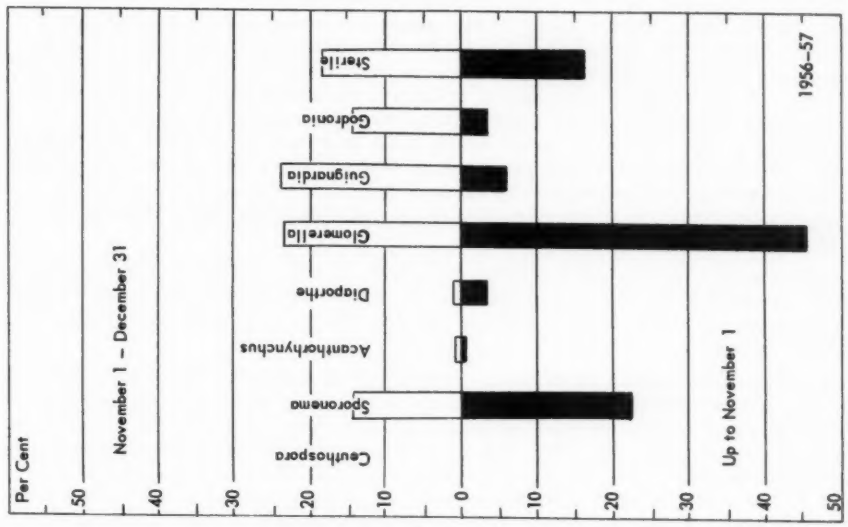

FIGURE 2. Comparison of frequency of occurrence of fungi before and after November 1 in spoiled Howes, Early Black, and McFarlin berries from Massachusetts during the years 1956 and 1957.

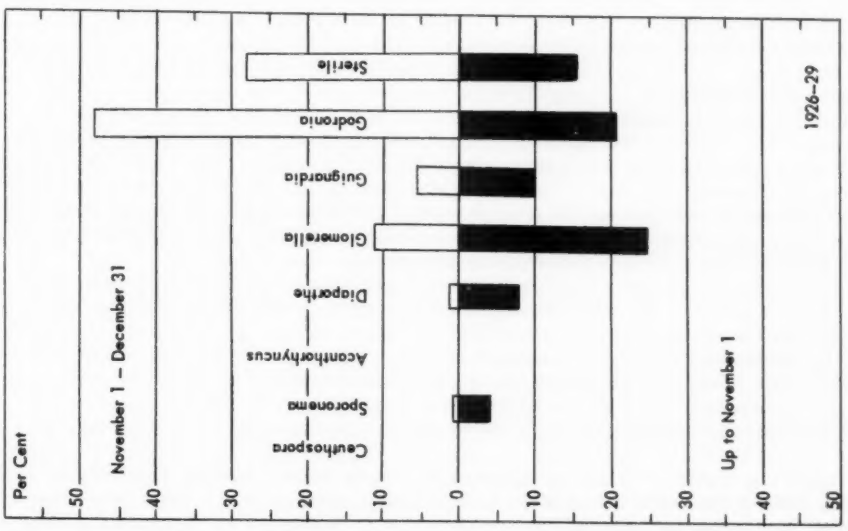

FIGURE 1. Comparison of frequency of occurrence of fungi before and after November 1 in spoiled Howes and McFarlin berries from Massachusetts during the years 1926 to 1929 inclusive.

based on 914 determinations made during the period of August through December, 1956-1957. (see Fig. 3).

In 1956-1957 Sporonema was second in importance as a fruit rot fungus and was recovered more often from Howes than from Early Blacks. Thus it appears to have assumed quite a different role since the previous studies. Glomerella was much more prevalent on Early Blacks than on Howes in 1956; however, in 1957 it was recovered from berries of each of these varieties in approximately the same ratio. Guignardia, which was recorded as an important early rot on Howes in 1926-1929, was more prevalent on Early Blacks in 1956-1957. However, since there is no record of Guignardia isolates in the 1926-1929 studies, these results cannot be compared. In 1956 Godronia caused a significant amount of rot of Howes; however, in 1957 it was relatively scarce. Rudolph and Franklin (2) noted a similar relationship of Godronia to Howes, for in 1916 it was abundant and in the following year of much less importance. Diaporthe, which in past years was recorded as being a fairly important early rot in Early Blacks, was of very minor importance in 1956-1957.

The following observations are made in regard to the relation of the rot pathogens to Early Blacks and Howes:

1. Sporonema is a more important cause of rot in both of these varieties than during the previous years in which studies have been made. In contrast to the earlier studies which reported that Sporonema occurred mainly on Early Blacks, the current findings were that it was more abundant on Howes.

2. There is considerable variation in regard to the type of fungus which causes significant amounts of spoilage to these two varieties in different years. As an example, Glomerella, which was reported as occurring most often on Howes in 1916-1917 and 1926-1929, was more prevalent on Early Blacks in 1956. In 1957, however, it occurred on Early Blacks and Howes in about the same proportions.

3. If Early Blacks and Howes are at all differentially resistant to the rot pathogens, it is only to a very small degree.

Relative Abundance of Fungi and the Relation of Keeping Quality on "Early Water" as Compared with "Late Water" Bogs

Cranberry bogs are frequently held under water from the middle part of April to late in May. This practice delays the bloom of the cranberry flowers approximately 2 weeks and is known in the industry as "late water", as opposed to "early water" in which the winter flood is drawn from the bog during March or early April.

Experimental evidence indicates that infection of the cranberry fruit by rot organisms takes place through the receptive stigma of the flower. Consequently, any cultural practice that alters the time of bloom could conceivably affect the host-parasite relationship in a given year. For example, if the period during mid-June when the "early water" bogs are in bloom is dry, and the reverse is true during the first weeks of July when the "late water" bogs are in bloom, one would expect that the "late water" bogs would be of poorer quality that year. This study was made for the purpose of examining this theory.

Table 2 gives the receptive dates for "early water" and "late water" Early Black cranberry flowers on the State Bog, East Wareham, Massachusetts, in 1956 and 1957, and the number of periods that were thought to be favorable for infection during this time. Conditions favorable for infection were defined as a period in which rain occurred, and the relative humidity beneath the vine cover was 90 percent or greater for periods of 18 hours or more. In June 1956, as infection period of 50 hours was recorded, and in July 1957, one of 48 hours occurred, but generally the infection periods did not exceed 24 hours during the 2 years of this study.

From the figures in Table 2, it was surmised that "early water" berries should have been of good quality in 1957 if favorable periods for infection during bloom were the prime factors related to quality. Quite the opposite was true; many lots of "early water" berries did not hold up in storage. "Late water" berries behaved in the same manner.

Table 3 gives the relationship of field and storage rot in "early water" and "late water" Early Blacks on bogs in Eastern Massachusetts in 1956-1957. A storage period of 6 weeks represents an attempt to arrive at a useful average, since cranberries processed for the fresh fruit market may be shipped in a few days or held in common storage for 3 months prior to

Table 2. Dates at which "early water" and "late water" Early Black cranberry blossoms were receptive in 1956-1957, and the number of infection periods which occurred during this time interval at the State Bog, East Wareham, Massachusetts.

Year	Early water	Number of infection periods	Late water	Number of infection periods
1956	June 22 - July 12	5	July 8 - July 26	2
1957	June 13 - July 26	0	June 24 - July 10	3

Table 3. Field and storage rot of Early Blacks in Massachusetts, 1956 - 1957.

Year	Number of bogs sampled	Water management	Field rot[a] (percent)	Storage rot[b] (percent)	Total loss (percent)
1956	14	Early water	3.8	5.5	9.3
	11	Late water	2.4	5.8	8.2
1957	18	Early water	4.0	10.2	14.2
	4	Late water	2.1	9.6	11.7

[a] Percent figures represent averages of all samples.
[b] Storage periods were for 6 weeks following harvest.

shipping. Samples were a minimum of 500 berries in size, and all were taken from bogs not treated with fungicides.

Though there were more infection periods in 1956 than in 1957 (Table 2), cranberries were generally of better quality in 1956 than in 1957 (Table 3). "Late water" berries appeared to be of slightly better quality in both years (this difference is not statistically significant), despite the fact that the conditions for infection during the late bloom period in 1957 apparently were more favorable than during the early bloom period.

Figure 4 gives the occurrence of the six most important cranberry rot fungi on "early water" and "late water" cranberries in 1956-1957. These figues are based on 977 determinations made during the periods of August through December of these years. Glomerella occurred most often on "early water" berries in both of these years, and was most prevalent on "late water" berries in 1957. Sporonema was by far the most important fungus on "late water" berries in 1956, and was second to Glomerella on "late water" berries in 1957 and on "early water" berries in 1957. It was interesting to note that while Godronia was important on "early water" berries in 1956, it was more important on "late water" berries in 1957.

Observations on the interrelationships of rot fungi "early water" and "late water" holding, and the period of bloom are as follows:

1. The number of infection periods during bloom appears to be unrelated to the abundance of specific rot fungi in spoiled cranberries at and following harvest. In 1957 there were no periods (as defined in this paper) which favored infection during the "early water" time of bloom, as opposed to 1956 when five such periods were recorded (Table 2). Despite this, Glomerella, Sporonema and Guignardia were relatively more abundant on "early water" berries in 1957 than they were the previous year (Figure 4).

It is probable that conditions affecting the incubation of rot fungi within the berries are more directly related to cranberry spoilage than the quantitative amount of infection. There is much evidence to support this view. In a study carried out in October and November 1957, rot organisms were isolated from 73.4 percent of the berries which apparently were sound at the time cultures were made (Zuckerman, unpublished data). Wilcox (5) wrote that summer temperatures, water management practices, commercial methods of harvesting and storing the fruit, etc., have significant effects on quality. This theory is also supported by evidence that cranberry rots do not spread from berry to berry in storage (1). Therefore, it appears

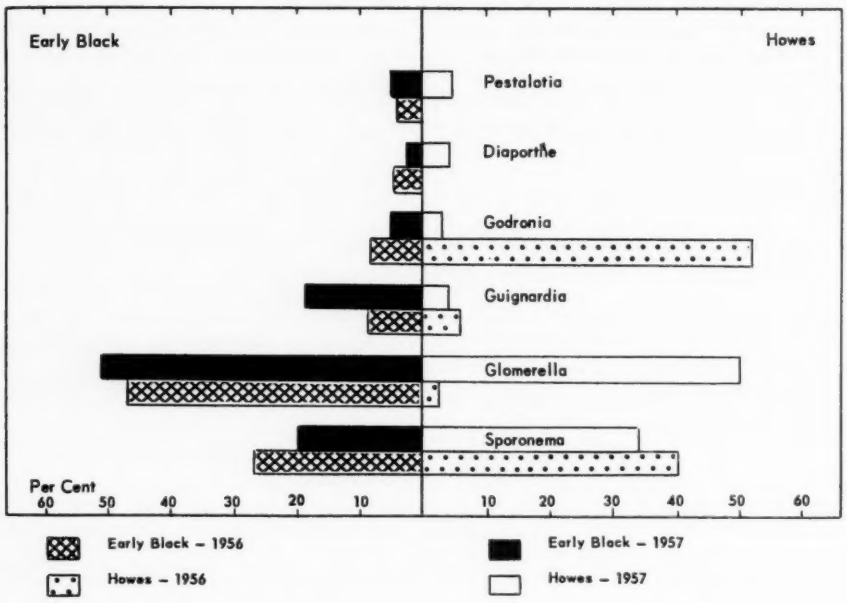

FIGURE 3. Frequency of occurrence of fungi in spoiled Howes as compared with Early Black berries from Massachusetts during the years 1956 and 1957.

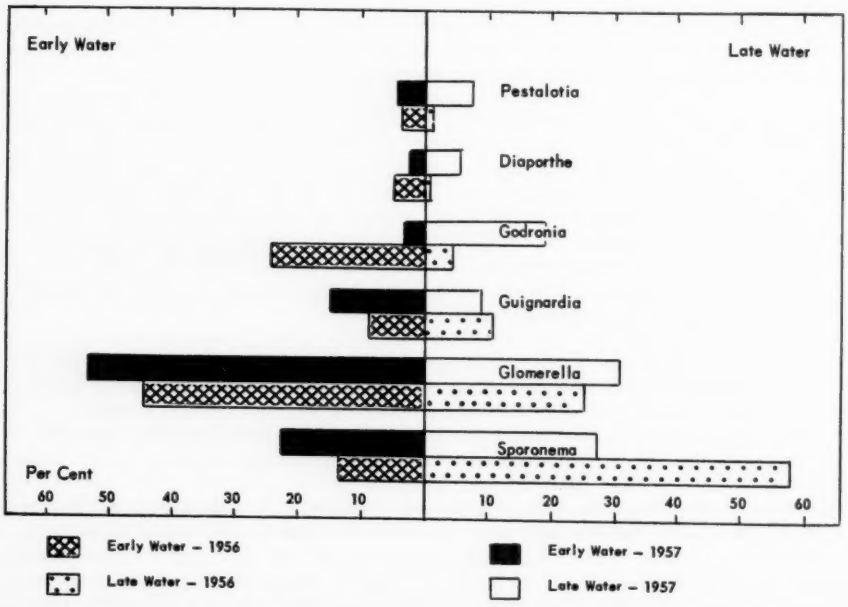

FIGURE 4. Frequency of occurrence of fungi in spoiled berries from "early water" bogs as compared with "late water" bogs in Massachusetts during the years 1956 to 1957.

that a great percentage of cranberries contain rot fungi at harvest, but that they do not break down unless the proper stimulus or "triggering action" occurs.

2. The theory that "triggering conditions" rather than infection conditions are of prime importance in cranberry spoilage is given indirect support by the observation that although there were more infection periods in 1956 than in 1957, the cranberries were of better quality in 1956 than in the following year. These berries were harvested from bogs where the normal infection pattern had not been disturbed by spraying with fungicides. Of possible significance is the fact that temperatures in eastern Massachusetts averaged 3 degrees below normal in September 1956, whereas in September 1957 they averaged 3 degrees above normal.

A Study of External Symptoms of Fruit Rot as Related to the Causal Organism

Shear et al. (3) state that the external symptoms of the cranberry fruit rots are so similar that it is impossible to distinguish them without making artificial cultures. In the study reported in this paper, a record was kept of the external symptoms for each berry cultured. When a culture was identified, these macroscopic symptoms were entered on the index card with other data pertaining to the culture. The symptoms of cranberry fruit rot, which are quite varied, were divided into 33 categories for the purposes of this study.

The results of this portion of the work confirm the conclusions of Shear et al. (3) to the effect that cranberry rots cannot be distinguished without resorting to artificial cultures.

CONCLUSIONS

On the basis of the data presented in this paper it is apparent that Sporonema has currently assumed a position of importance in the spoilage picture in Massachusetts, whereas at the time of the previous studies it was not a serious parasite on this crop. Whether this relationship will remain fixed in the future can be gauged only by continued study. It is possible that the current upsurge of Sporonema may be related to cultural practices aimed at increasing the crop per unit area, but experimental evidence in support of this theory is lacking. On Early Blacks or Howes, and with "early water" or "late water", Sporonema was one of the primary despoilers of Massachusetts cranberries during the past 2 years. In contrast, in the 1916-1917 and 1926-1929 studies it was considered least important of the fungi generally found in spoiled cranberries.

Glomerella remains, as in the past, the single most important rot organism in Massachusetts.

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DEPARTMENT OF ENTOMOLOGY AND PLANT PATHOLOGY, CRANBERRY EXPERIMENT STATION, UNIVERSITY OF MASSACHUSETTS, EAST WAREHAM, MASSACHUSETTS

POLLEN TRANSMISSION OF NECROTIC RING SPOT VIRUS IN CHERRY[1]

R. D. Way and R. M. Gilmer

Summary

Five of 18 cherry seedlings that resulted from a cross of healthy English Morello (female parent) by virus-infected Montmorency (male parent) were found to be infected with necrotic ring spot virus when indexed on cucumber seedlings. Fifteen cherry seedlings resulting from a cross of healthy English Morello (female parent) by healthy Montmorency (male parent) all indexed virus-free. The possibility of transmission of necrotic ring spot virus to healthy trees through the agency of infected pollen and the significance of such transmission in a virus control program are discussed.

Transmission of necrotic ring spot virus (NRSV) and sour cherry yellows virus (SCYV) through the seeds of several species of Prunus has been established by several workers (2, 3, 4, 5, 7, 8). The actual mechanism of virus transfer to seeds has, however, not been ascertained. It has been tacitly assumed that seed infection resulted from virus transfer from the female parent. Cochran (4), in establishing the occurrence of seed transmission of ring spot virus in mazzard (P. avium L.) seeds, stated: "The amount of seed ..... coming from trees infected with ring spot is not known, nor is the percentage of ring spot which would result if the seed were taken from a single infected tree." In testing transmission of ring spot virus in peach seeds, Cochran (5) obtained seeds directly from an infected female parent. Cation (2, 3), in transmission tests with mahaleb (P. mahaleb L.) and Montmorency cherry seeds, similarly harvested seeds from infected female parents.

Ehlers and Moore (6) have recently shown that pollen from virus-infected sour cherry trees carried several of the Prunus viruses (viruses A, B, E, and, possibly G)[2]. This fact suggested that seed infection might result from fertilization with virus-infected pollen. Moreover, Callahan (1) found elm mosaic virus to be transmitted to seeds from virus-infected pollen; he reported that an average of 30.5 percent of 1523 elm seedlings resulting from four crosses of virus-free ovules with virus-infected pollen were infected.

The present paper presents evidence that NRSV is transmitted by infected pollen to seeds developed on healthy trees.

EXPERIMENTATION

In 1954 flowers on selected branches of four virus-free English Morello cherry trees were emasculated at the balloon stage of development to prevent self-pollination or cross-pollination by insects. Pollen from Montmorency cherry trees was applied to the stigmas of the emasculated flowers 2 to 5 days after emasculation. The Montmorency pollen was collected from four trees with the following virus status: 1 tree virus-free, 1 tree infected with NRSV, and 2 trees infected with NRSV and SCYV. The virus status of the Montmorency pollinators had been determined in 1951 and 1952 by indexing each tree on an appropriate indicator host range.

Seedlings resulting from these crosses were grown in the greenhouse in the summer of 1955. In May 1956, they were transplanted to the field in an area isolated from other Prunus species by at least 300 feet. The seedlings received the normal schedule of sprays in 1956 and 1957 to control insects and fungus diseases. None of them bloomed in 1956 or 1957.

On April 28, 1958 each seedling was indexed for possible virus content on National Pickling cucumber seedlings, using the methods described by Gilmer et al. (9). Results of indexing are summarized in Table 1.

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[1] Approved by the Director as Journal Paper No. 1139, New York State Agricultural Experiment Station, Geneva, New York.

[2] Virus A = NRSV, virus B = SCYV (Posnette et al. (10)). Virus G is probably a strain of virus A (Fulton (7)).

Table 1. Transmission of NRSV to seedlings of a cross of healthy English Morello x infected Montmorency.

Virus status of English Morello seed parent	Virus status of Montmorency pollen parent	Seedlings indexed (Number)	Seedlings infected (Number)
Virus-free	Virus-free	15	0
Virus-free	Infected[a]	18	5

[a] Infected pollen source trees contained either NRSV or NRSV + SCYV.

No specific attempt was made to identify the actual virus or viruses transmitted to cucumber seedlings, but the reaction of the cucumber indicator plants, necrosis of the apical bud, yellow mottle, and rapid collapse, was characteristic of the reaction caused by infection with NRSV. Other viruses may also have been present but were not detected because of masking of symptoms in the presence of symptoms incited by NRSV.

DISCUSSION

The possibility of NRSV transmission by agencies other than pollen is not indisputably disproved by this study, since the seedlings were not indexed until they had grown two seasons in the field. That transmission of NRSV by agencies other than pollen did occur seems highly unlikely, since none of the seedlings of the crosses made with both parents virus-free contained virus while all of the seedlings in which virus was found had an infected pollen parent.

Two important implications in the control of NRSV are suggested by this study. First, if NRSV is transmitted through pollen to seeds developed on virus-free trees, it seems equally possible that NRSV could be translocated from the developing seeds back into the mother tree. The latter point has not been experimentally established in this study, but pollen transmission followed by transfer of the virus back into the seed tree affords a very plausible explanation of the sudden rapid spread of NRSV in young virus-free orchards during the period 5 to 8 years after planting. A comparatively slow spread of NRSV in such young orchards frequently first occurs in trees in the outer rows of the orchard, and is followed by extremely rapid spread of the virus through the remainder of the orchard 2 to 3 years after its initial appearance. It is during the period 5 to 8 years after planting that heavy bloom and fruit set begin to occur.

Second, virus-infected understocks are the principal source of infection of cherry nursery trees in New York (9). In certified plantings propagated with virus-free budwood, about 5 to 7 percent of the nursery trees are virus-infected as a result of propagation on infected seedling understocks. A concerted effort is presently being made in New York and on the Pacific Coast to eliminate infected understock seed source trees. Pollen transmission of NRSV further complicates this control program, since seeds, even though produced on a virus-free tree, may have been infected from the pollen parent.

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DEPARTMENTS OF POMOLOGY AND PLANT PATHOLOGY, NEW YORK STATE AGRICULTURAL EXPERIMENT STATION, GENEVA, NEW YORK

PRESENT STATUS OF INFORMATION ON HOST RANGE AND HOST REACTIONS TO PEACH MOSAIC VIRUS

L. C. Cochran and T. S. Pine[1]

A knowledge of the species and varieties of plants susceptible to infection by the peach mosaic virus is paramount to the development and maintenance of quarantines designed to prevent movement of that virus from infected to noninfected areas. In addition, it is necessary to recognize the disease on hosts which express symptoms and to have suitable test procedures for determining the presence of the mosaic virus in hosts on which symptoms are not expressed. Most Prunus species and varieties are vegetatively propagated, either as rooted cuttings or from buds or scions; hence, should the parent stocks become infected, propagations from them are almost certain to be infected. Numerous incidents of viruses being carried in infected budwood and nursery trees are on record; with little doubt, the present occurrence of peach mosaic traces to its distribution in infected nursery material before quarantines were initiated.

SUSCEPTIBLE SPECIES

Determination of susceptibility of host species to the mosaic virus was based on either natural infection of the host suspect, as evidenced by transmission to healthy peach, or infection as a result of experimental inoculation. Species experimentally inoculated were grown on their own roots as seedlings or as rooted cuttings and were graft-inoculated with mosaic-infected peach tissue. Inoculation was considered adequate if the peach inoculum was alive 1 month after the graft was made. In all cases, indexing of the inoculated suspects to healthy peach was attempted during the following growing season regardless of whether the suspect showed mosaic leaf symptoms.

Prunus americana -- American plum [2,3,4]
P. amygdalus -- Almond [2,3]
P. angustifolia -- Chickasaw plum [2,3,4]
P. armeniaca -- Apricot [2,3,4]
P. armeniaca var. ansu -- Ansu apricot[2]
P. besseyi -- Bessey cherry [3]
P. bokhariensis -- Bokar plum [2]
P. cerasifera -- Myrobalan plum [2,3]
P. davidiana -- David peach [2,3]
P. domestica -- European or domestica plum[2, 3, 4]
P. hortulana -- Hortulan plum [2,3,4]
P. insititia -- Damson plum [2,3]
P. japonica -- Korean cherry [3]
P. mexicana -- Mexican plum [2,3]
P. mume -- Japanese apricot [2,3]
P. munsoniana -- Pottawattamie plum[2, 3, 4]
P. persica -- Peach (all varieties of approx. 200 tested)[2, 3]
P. persica var. nectarina -- Nectarine (all varieties tested)[2, 3]
P. reverchonii -- Hog plum [2]
P. salicina -- Japanese plum [2,3,4]
P. subcordata -- Pacific plum [3]
P. tangutica -- Tangut plum [2]
P. tomentosa -- Manchu cherry [2,3]
P. umbellata -- Flatwoods plum [2]

IMMUNE SPECIES

Host species inoculated and indexed 1 year later by budding from them to peach without production of characteristic symptoms are considered immune. The following species were tested and are considered immune:

Prunus avium -- Sweet cherry
P. cerasus -- Sour cherry
P. emarginata -- Bitter cherry
P. fasciculata -- Desert almond

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[1]Formerly Plant Pathologist, and Plant Pathologist, Crops Research Division, Agricultural Research Service, U. S. Department of Agriculture, cooperative with the California Agricultural Experiment Station, Riverside, California.
[2]Found naturally infected.
[3]Experimentally infected.
[4]Expressed symptoms only with certain virus strains or on certain varieties.

P. fremontii -- Desert apricot
P. ilicifolia -- Hollyleaf cherry
P. mahaleb -- Mahaleb cherry
P. pensylvanica -- Pin cherry
P. serotina -- Black cherry
P. virginiana var. demissa -- Western chokecherry
Rosa odorata -- Tea rose
Pyrus malus -- Apple

SUSCEPTIBILITY AND REACTION OF CHERRIES

All the horticultural varieties of sweet and sour cherries and most of the so-called "wild cherry" species that have been tested are immune. Prunus besseyi (Bessey or sand cherry), P. tomentosa (Manchu cherry), and P. japonica (Korean cherry) are exceptions; all three were experimentally infected and developed characteristic leaf mottling. That they are more closely related to peach than the other cherries is evidenced by the fact that they are graft-compatible with peach, whereas the other cherries will make organic union but fail to produce shoot growth. The Bessey and Manchu cherries hybridize with P. cerasifera and P. americana; thus they should perhaps be more properly considered plums rather than cherries.

SUSCEPTIBILITY AND REACTION OF PLUMS

The native or naturalized "wild" plums of the South and Southwest appear to be excellent hosts of the peach mosaic virus and may have been the natural reservoir from which original spread to peach took place. The chickasaw, American, and pottawattamie are thicket plums that produce sucker shoots. The wide distribution of peach mosaic in the Rio Grande and Colorado River Valleys undoubtedly traces to the carrying of shoots of infected plums from one locality to another by natives who prized them for their fruits. Some strains of the peach mosaic virus produce mottled patterns on the leaves of these plums, but others infect them without symptoms. American plum usually expresses more pronounced symptoms than does pottawattamie or chickasaw. Prunus subcordata is susceptible to the peach mosaic virus; thus, should the virus escape to the Pacific Northwest where this plum is native and where named varieties of it are grown commercially, it might provide a natural reservoir from which spread could take place to peaches and apricots.

Natural spread from pottawattamie plum to peach was demonstrated in an orchard near Moab, Utah[5]. The annual recurrence of mosaic in replanted peaches near a dooryard hedge of pottawattamie plums pointed to the hedge as a source from which spread was taking place. All peach trees were removed from the property and 50 young peach trees were planted in orchard formation near the hedge during the following winter. Of the 45 trees which survived, 19 showed symptoms of peach mosaic the following spring. The plum hedge row showed no symptoms but was shown by graft indexing to peach to be 100 percent infected. The hedge row was destroyed and the mosaic-affected peach trees were removed. The orchard was replanted, and none of the 50 trees which survived until the following spring were mosaic-infected.

Several ornamental plum species are being widely propagated by the nursery trade, but most of these have not been adequately tested to determine their status as peach mosaic hosts. Prunus cistena, a hybrid between P. pumila (a close relative of P. besseyi) and P. cerasifera var. atropurpurea, is a new ornamental plant in the nursery trade and is under test. It should be regarded with suspicion, however, since both of its parents are known to be susceptible to the peach mosaic virus. The common purple-leaved plums are closely related to P. cerasifera (myrobalan plum) and should be regarded with suspicion.

The horticultural varieties of plums have been difficult to study. Only occasional trees of a few varieties (Becky Smith, Cain Seedling, Flaming Delicious, Damson, and French prune) have been found naturally infected in orchards. Emelie and Giant prune check trees growing in the experimental plots contracted mosaic as a result of natural spread. Trees in many orchards of Japanese and domestica-type plums interplanted with peach, in which nearly 100 percent of the peaches were mosaic-affected, were indexed without finding natural infec-

[5]This work was in cooperation with the Plant Pest Control Division of the Agricultural Research Service and the Utah State Department of Agriculture.

tion in the plums[6]. These same varieties of plum growing on peach or marianna plum rootstocks are susceptible to infection when graft-inoculated. Work is now under way to determine which horticultural plum varieties are susceptible when grown on their own roots. After inoculation with certain strains of the peach mosaic virus, the following plum varieties produced varied amounts of leaf symptoms: French, Standard, Giant, Green Gage, Tragedy, Beauty, Shiro, Damson, Becky Smith, Cain Seedling, and Flaming Delicious. It is presumed that varieties which show leaf symptoms will support infection and therefore are susceptible hosts to that strain of the virus.

SUSCEPTIBILITY AND REACTION OF PEACH AND NECTARINE VARIETIES

All peach and nectarine varieties appear to be equally susceptible to infection by the peach mosaic virus, either as a result of natural spread or from graft inoculation. However, they vary in severity of symptom expression. Most freestone varieties are severely damaged, but most of the clingstones tolerate the virus with little damage. The use of tolerant varieties in areas where roguing of infected trees is used as a control measure is undesirable because of greater difficulty of diagnosis, especially where the "milder" virus strains are present. On the other hand, tolerant varieties are desirable in areas where roguing is not feasible and peach growers must operate in the presence of the disease.

Peach and nectarine varieties have been studied for their relative reaction to peach mosaic. Ten trees of each variety were propagated on Lovell peach seedling rootstock and grown 4 feet apart in nursery rows for 1 year. Two trees of each were inoculated from a stock culture of peach mosaic virus known to produce moderately severe symptoms on peach but no symptoms on apricot. Two additional trees were inoculated with another stock culture which produces severe symptoms on both peach and Royal apricot. Observations on most of the varieties were made during two growing seasons and the varieties were catalogued in three classes according to their relative response.

Peach Varieties Grouped According to their Relative Reaction to Peach Mosaic

Severely Damaged Varieties

Afterglow	Frank	Kirkman Gem
Alamar	Fredberta	Late Champion
Ambergem	Freedom (nect.)	Late Elberta
Babdon	Gage Elberta	Le Grand (nect.)
Belle	Gold Dust	Loring
Blazing Gold	Gold Medal	Maybel (nect.)
Bonita	Golden Blush	Maygold
Candoka	Golden Heath	Meadow Lark
Chaffey	Golden State	Merrill 49'er
Colora	Goldeneast	Merrill Aurora
Dixigem	Goldenglobe	Merrill Beauty
Dixired	Halberta	Merrill Brilliant
Donwell	Hale Harrison Brilliant	Merrill Dandy
Duke Hale	Hardy Berta	Merrill Delicious
Early Elberta	Hiland	Merrill Goldrush
Early Halehaven	Hiley	Merrill Hale
Early Rose	Illinois	Merrill June
Elberta	Impon	Merrill Late Canner
Fairhaven	J. H. Hale	Merrill Late Gold
Fay Elberta	July Hale	Merrill Rodeo
Fertile Hale	Kalhaven	Nectar
Fireglow	Kette	Newboy (nect.)
Flamingo	King Tut	Newday

[6]Hutchins, Lee M., E. W. Bodine, L. C. Cochran, and Gilbert L. Stout. 1951. Peach mosaic. In Virus Diseases and Other Disorders with Virus-like Symptoms of Stone Fruits in North America. U. S. Dept. Agr. Agr. Handb. 10. pp. 26-36.

New Jersey State Rose
Pacemaker
Philp (nect.)
Prairie Dawn
Prairie Rambler
Quetta (nect.)
Ranger
Raritan Rose
Redcap
Redhaven
Reisner's Improved Rio Oso Gem
Rio Oso Gem
Roberta
Shimm's Delicious
Shippers Late Red
Silverlode (nect.)
Spotlight
Starking Delicious
Sullivan Elberta
Summercrest
Summer Rose
Sunday Elberta
Sunhigh
Triogem
Vanguard
Vedette
Veteran
Veter's (Early Elberta)
Welberta
White Hale
Wilma

Medium Damaged Varieties

Admiral Dewey
Alexander
Augbert
Babcock
Brackett
Buttercup
Cameron
Cardinal
Carman
Champion
Cornet
Corona*
Cumberland
Curlew
Dripstone
Early Babcock
Early East
Early Wheeler
Eclipse
Fair Beauty
Fortuna*
Frostqueen
Garden State (nect.)
Gloribloom
Golden Jubilee
Halate
Halegold
Halehaven
Harter*
Hauss*
Herb Hale
Jerseyland
John Rivers (nect.)
July Elberta
Krummel
Lippiot's Late (nect.)
Lizzie
Lucas Beauty
M. A. Blake
Massasoit
Maydon
Maywell
Merrill Gem*
Merrill Yellow King
Michigold
Ozark
Penryn
Pioneer (nect.)
Prairie Clipper
Prairie Daybreak
Prairie Schooner
Prairie Sunrise
Ramsey
Redelberta
Redglobe
Redskin
Robin
Rochester
Rose Bud
Salberta
Sandhill No. 1
Solo
Southland
Stanwick (nect.)
Sunbeam
Sunday
Sungold
Tulip
Vaughn
Veefreeze
Vesper
Vimy
Wahlbert
Weldon
Western Pride
Wild Rose
Wiser*
Zarn

Slightly Damaged Varieties

Amador
Andora*
Australian Saucer
Carolyn*
Cortez*
Curry
Deberard*
Erly-Red-Fre
Fidler
Fisher
Gaume*
Greensboro
Halford No. 2*
Heath*
Johnson*
Lukens
Marigold (nect.)
Mikado
Nestor
Orange Cling*
Paloro*
Panamint (nect.)
Peak*
Phillips*
Redwing
Shasta*
Sims*
Springtime
Stanford*
Stuart*
Sullivan No. 1*
Sullivan No. 2*
Sullivan No. 4*
Tudor*
Tuskena (Tuscan)*
Valiant
Vivian*
Walton*
Williams*
Zuni

*Clingstone

CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, ORLANDO, FLORIDA

PRELIMINARY STUDIES ON YELLOW-LEAF OF OATS IN GEORGIA

1. AN APHID TRANSMISSIBLE VIRUS[1]

Luther L. Farrar

During the last 4 years a syndrome appeared, primarily on Victorgrain oats, in several counties in Georgia (3, 4). During the 1953-54 and 1955-56 seasons the malady was observed in small amounts and in scattered locations[2]. In 1956-57 it was widespread and caused considerable loss. However, in 1957-58 it was again sporadic and occurred in small amounts. A similar occurrence has been reported from South Carolina (1, 2). At first it was reported (1) that a species of Helminthosporium could cause oat chlorosis but later (2) it was stated that the cause is unknown. Results of field, laboratory, and greenhouse studies are presented in an effort to combine observations pertaining to this potentially serious malady of oats.

SYMPTOMS

The most striking symptom is a golden yellowing which occurs in (a) somewhat circular patches from 2 to 30 feet in diameter (Figure 1), (b) irregular patches, (c) scattered plants, and (d) occasionally entire fields of 15 to 30 acres. However, apparently healthy plants are observed in the affected areas. On close macroscopic examination the following symptoms can be observed: (a) longitudinal yellow striping of leaves in the areas between the veins (Figure 2), (b) a golden yellowing of entire leaves on the upper portion of plants (Figure 3), (c) a golden yellowing of the entire central portion of leaves, that is, only the leaf tip area and the area anterior to the ligule are light to dark green (Figure 2), (d) twisting and curling of leaves, (e) chlorosis of oat heads emerging from the boot, (f) blasting of florets, (g) stunting of plants (Figure 4), (h) reduced panicle size, and (i) usually a severe reduction in yield.

MATERIALS AND METHODS

Yellow-leaf affected and apparently healthy plants were brought to the laboratory for comparison and microscopic examinations. Sections of apically and basally affected leaves were plated on oatmeal agar and root sections from both yellow-leaf affected and apparently healthy Victorgrain plants were washed in running tap water for 12 to 16 hours and plated on water agar using standard procedures. Leaf sections were dechlorophyllized by boiling in 1N potassium hydroxide for microscopic examination.

Tests were run on soil samples taken at random from both yellow-leaf affected and apparently healthy Victorgrain oat areas. In addition, the minor elements boron, iron, magnesium, manganese, and molybdenum and a minor element mixture were applied to random plots of yellow-leaf affected plants[3].

Soil and root samples were collected from both yellow-leaf affected and apparently healthy areas and examined for plant parasitic nematodes.

Field notes were taken in yellow-leaf areas on: topography, field location, fertilizer applied, dates of fertilization, planting dates, aphid population, and on plots treated with an insecticide (Thimet).

Essentially three methods were used to restrict aphid feeding to the desired plant. In 1957 large plastic cages (30 x 20 x 18 inches) were used to restrict apple grain aphids to virus free and yellow-leaf affected plants. In 1958 individual aphid cages were made by cutting 1 x 1/2-inch sections from sheet cork and cutting holes in the center with a number 2 cork borer. Sections of nylon hose were glued to these cork sections with Duco cement (Figure 5). These individual cages were fastened to leaves with two adjusted "silverette" gem paper clips (size No. 1). These paper clips were adjusted by placing them on a 4 x 1 x 1/4-inch section of sheet iron and shaping with a pair of pliers (Figure 6). This made the retaining sides 1/4 inch apart, and parallel (Figure 6), and resulted in equalized pressure across the individual cage and leaf. This

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[1] Contribution of the Department of Plant Pathology, Georgia Experiment Station, Experiment, Georgia, Journal Series Paper No. 341.

[2] Observed by Dr. U. R. Gore, Agronomist, Georgia Experiment Station, Experiment, Georgia.

[3] The soil tests and minor element treatments were, for the most part, conducted by Dr. O. E. Anderson, Soil Chemist, Georgia Experiment Station, Experiment, Georgia.

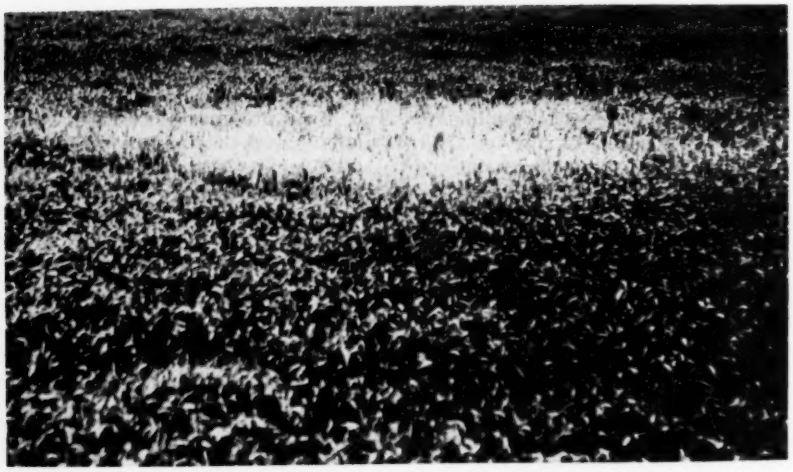

FIGURE 1. Typical patch of yellow-leaf in Victorgrain oats.

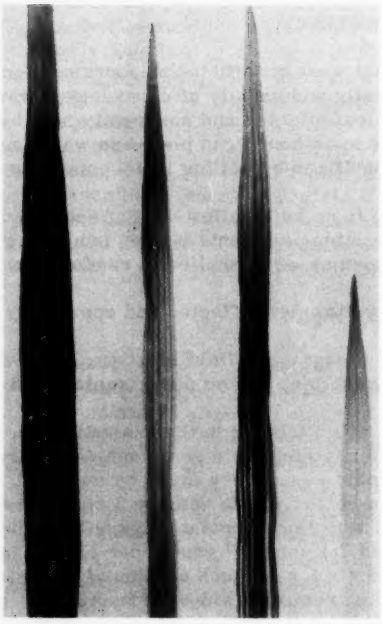

FIGURE 2. Typical striping and yellowing of Victorgrain leaves, check on left. Notice yellowing in center of leaf next to check, that is, only leaf tip and basal area green.

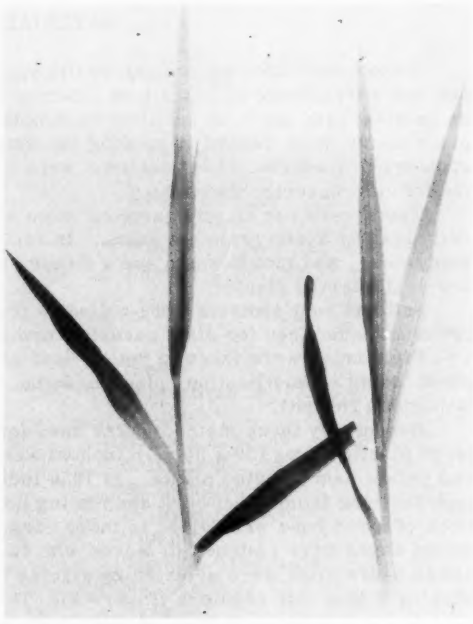

FIGURE 3. Typical striping symptoms on young tillers. Notice basal leaves are still dark green.

FIGURE 4. Stunting due to yellow-leaf. Victorgrain plants obtained from the same field.

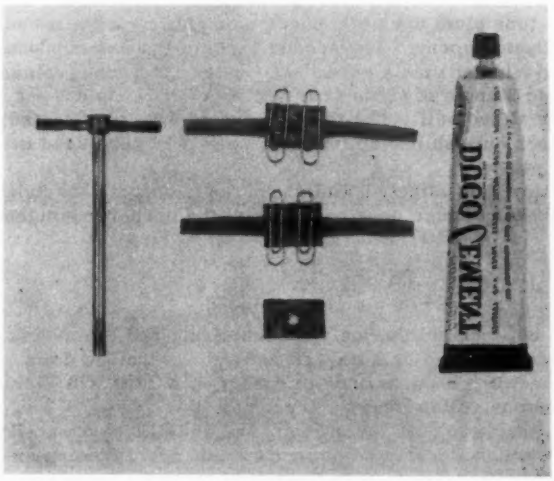

FIGURE 5. Individual aphid cages attached to leaves and principal items for construction.

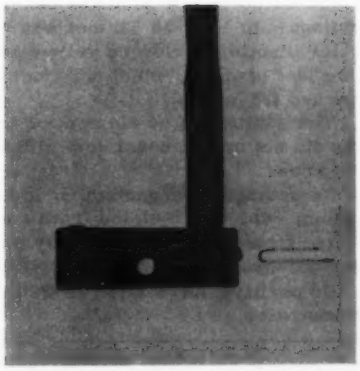

FIGURE 6. Pliers clamping gem clip to reshape it. Notice adjusted gem clip to left of sheet iron.

type cage was used to permit virus-free aphids to feed on yellow-leaf affected plants (Figure 8) and then the aphid and cage were transferred to virus-free plants growing under nylon hose (Figure 7). Thirdly, aphids were permitted to feed for 3 days on yellow-leaf plants under nylon hose and transferred with a moistened camel's-hair brush to virus-free plants growing under nylon hose.

Nylon hose was found to be very satisfactory when stretched over two inverted wire "U's" placed at right angles in the pot (Figure 7). The foot of the hose was clipped off and secured with a clothes pin and the top of the hose was kept tightly fastened to the top of the pots with a 1/2-inch rubber band (Figure 7). In this way the top of the cages could be lowered easily and aphids placed on, or taken off, the plants with a moistened camel's-hair brush. When using a camel's-hair brush a rolling motion was used to pick the aphid up on the side of the moist brush. A similar motion was used to deposit the aphid on a previously moistened leaf. It was found that less injury resulted to the aphids when the moistened brush was used in this manner and, also, the aphids adhered much better to a previously moistened leaf than to a dry leaf. Runs and small holes in the hose were sealed with Duco cement.

In 1957 virus-free apple grain aphids[4] were allowed to feed for 5 days on yellow-leaf affected plants. Then the aphids were transferred to virus-free Victorgrain plants (five plants per pot) growing in steam sterilized soil in 6-inch clay pots, where they were allowed to feed for 3 days before being killed by insecticidal spray. Appropriate checks were maintained. All plants were grown under plastic cages. In 1958 three tests were conducted[4]. In the first test, individual aphid cages were used to restrict single aphids to yellow-leaf affected leaves (Figure 8). The aphids were allowed to feed 3 days, then were transferred to virus-free Dubois oats growing in Vermiculite in 4-inch clay pots (one plant per pot), under nylon hose, and allowed to feed 3 more days before being killed with Systox spray. The second experiment was conducted in a similar manner, except that yellow-leaf affected plants were placed under nylon hose, aphids were placed on them and then transferred to 24 pots of virus-free Victorgrain (one plant per pot) growing under nylon hose in steam sterilized soil. The third experiment was conducted like the second except that eight pots of black hulless barley (one plant per pot) were used instead of Victorgrain oats for the indicator plants.

Juice was squeezed from leaves of yellow-leaf affected Victorgrain oats and used to inoculate healthy Victorgrain and Dubois oats growing under hose cages, using the carborundum leaf technique.

RESULTS

No positive correlation could be found between yellow-leaf affected plants and agronomic practices. In addition, yellow-leaf has been observed on both well and poorly drained soils.

In 1957 the disease was observed on an entire hillside of Victorgrain oats. In 1958 Victorgrain oats were planted to the same field and no yellow-leaf occurred.

Heavy aphid populations were observed early in both the 1956-57 and 1957-58 seasons. However, in the former season temperatures were moderate and oat growth and aphid feeding extended over a long period; whereas, the latter season was very cold and both oat growth and aphids were retarded until the first of March.

In Victorgrain clipping tests in 1956-57 and 1957-58, Thimet was applied to restrict aphids. In 1956-57 no yellow-leaf was observed in the clipped plots but was observed in surrounding plantings. In 1957-58 Thimet was applied after severe aphid infestations were observed in the earlier plantings. Severe yellow-leaf was observed in early plantings (Figure 9-A); however, in the last planting, which was just emerging when the Thimet was applied, no yellow-leaf was observed (Figure 9-B).

The addition of no element or minor element mixture tested affected the syndrome. Soil tests did not reveal consistent differences between yellow-leaf affected and apparently healthy plant areas.

No pathogenic organism could be consistently isolated from yellow-leaf affected leaves. *Pythium* sp. was isolated from roots of yellow-leaf affected plants. However, it could be isolated from roots of apparently healthy Victorgrain plants, also. These are essentially the same observations as those of Miller (5).

No positive correlation as to kind, or numbers, of nematodes with yellow-leaf could be demonstrated.

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[4] The apple grain aphids used in these tests were obtained from Dr. R. M. Endo, Pathologist, United States Department of Agriculture, University of Illinois, Urbana, Illinois.

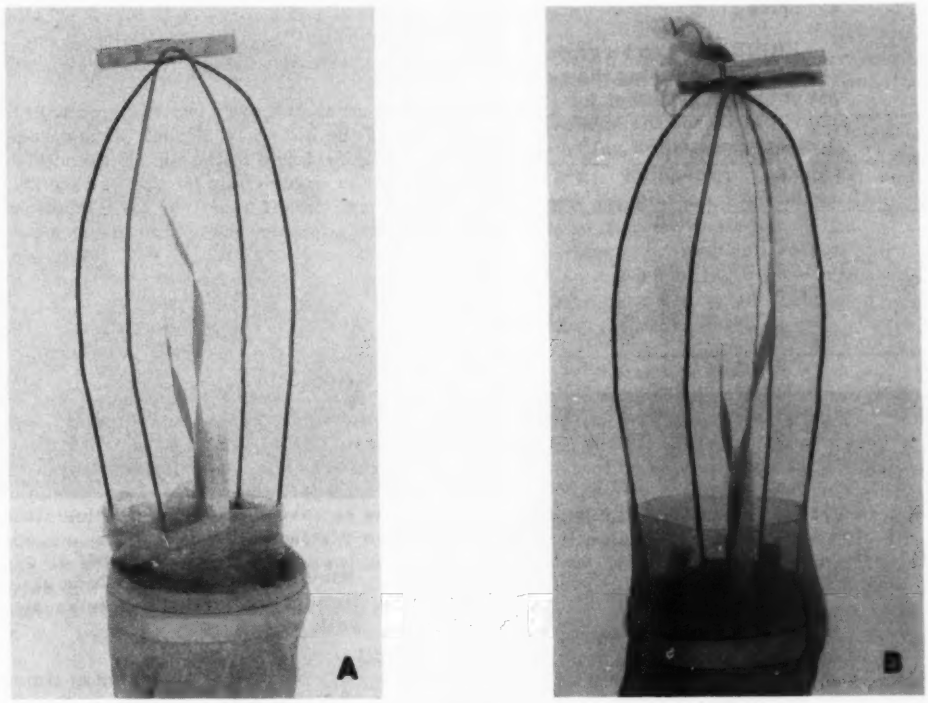


FIGURE 7. (Above). Use of nylon hose for aphid cages. A -- Hose down for placing aphids on plant. B -- Hose up for protection against undesirable aphid contaminations.

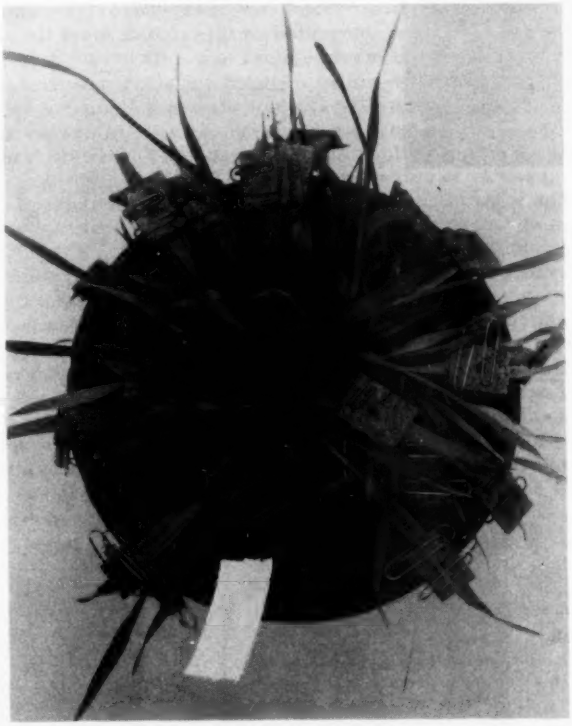
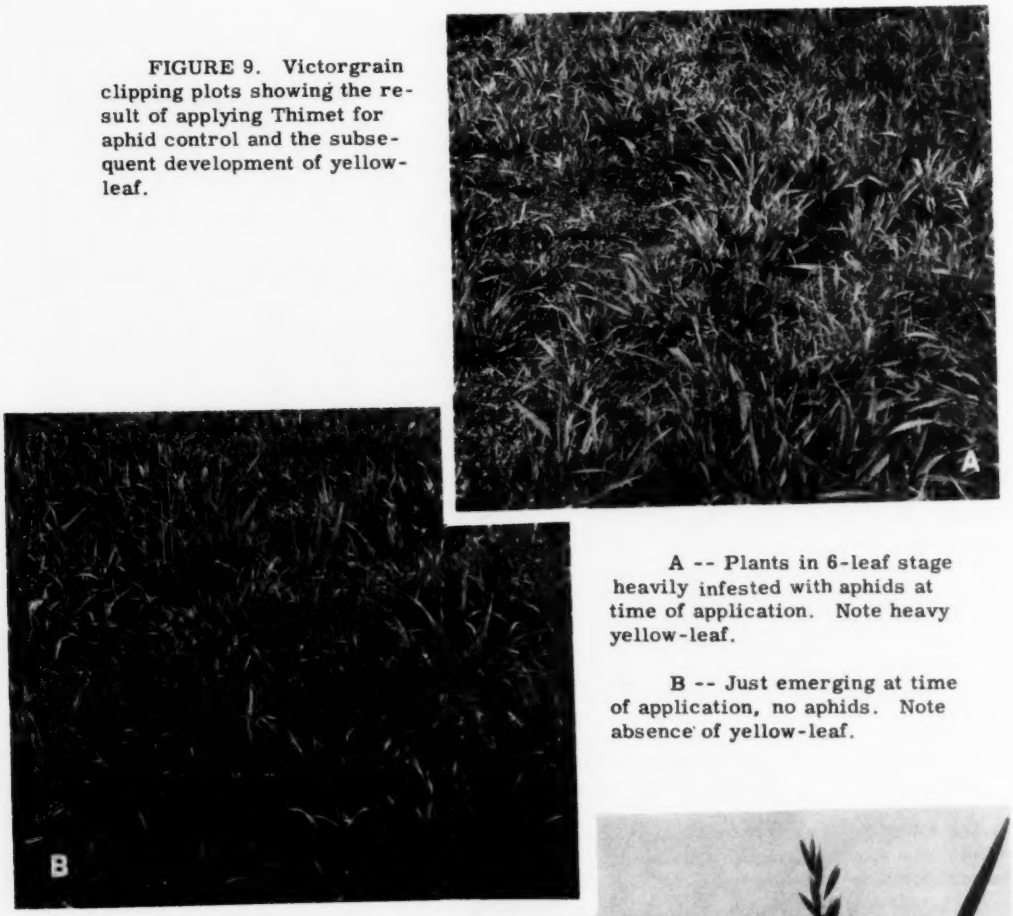


FIGURE 8. Individual aphid cages on the variety Victorgrain.

FIGURE 9. Victorgrain clipping plots showing the result of applying Thimet for aphid control and the subsequent development of yellow-leaf.



A -- Plants in 6-leaf stage heavily infested with aphids at time of application. Note heavy yellow-leaf.

B -- Just emerging at time of application, no aphids. Note absence of yellow-leaf.

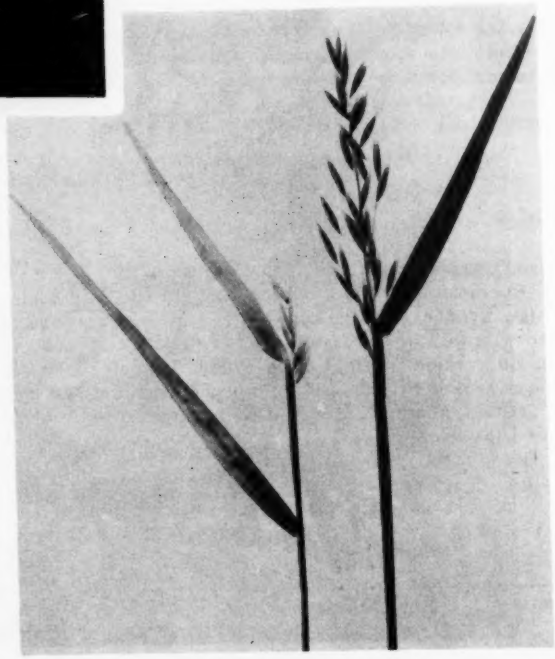


FIGURE 10. Reddish-brown mottling of upper leaves of Victorgrain due to yellow-leaf. Late striping occurred on lower leaves. Check on right.

In the 1957 preliminary aphid transmission test, leaves in each of the pots of Victorgrain inoculated, as described above, exhibited a yellow striping. No such striping occurred in checks (Table 1).

Table 1. Results of using apple grain aphids to transmit yellow-leaf to oats and barley.

Year	Variety	Inoculated		Non-inoculated	
		Number plants	Number infected	Number plants	Number infected
1957	Victorgrain oats	25	25	25	0
1958	Dubois oat	4	4	4	0
	Victorgrain oat	24	24	24	1
	Black hulless barley	8	6	16	0

In the 1958 tests all inoculated Dubois oats exhibited red-leaf symptoms in approximately 18 to 21 days, but not in controls. In the Victorgrain tests, plants exhibited a reddish-brown mottling of basal leaves in 14 to 21 days with yellow striping of upper leaves after 28 days. Frequently a reddish-brown mottling of upper leaves occurred (Figure 10), while lower leaves were striped. Only one plant in the controls showed these symptoms. This is attributed to aphid transmission in the greenhouse, as occurred with Oswald & Houston (7), after removal of the hose cages, since it appeared after 6 weeks (Table 1). Inoculated black hulless barley plants showed severe stunting after 2 weeks and yellow striped leaves after 3 weeks. Controls did not exhibit these symptoms (Table 1).

To date, all attempts to transmit this malady mechanically have been inconclusive.

DISCUSSION

Since (a) both striping of leaves and affected field areas similar to those reported by Oswald and Houston (6, 7) occurred on oats under Georgia conditions, apparent aphid transmission was obtained in all tests to date; (b) no pathogenic organism was consistently isolated from affected leaves; (c) Pythium sp. was isolated from both healthy and yellow-leaf affected plants; (d) there was no positive correlation between yellow-leaf affected areas and nematodes; (e) minor elements did not affect the syndrome; (f) yellow-leaf did not occur the succeeding year on the same land planted to Victorgrain; (g) yellow-leaf occurred on both well and poorly drained soils; and (h) observational data indicate that agronomic practices are not responsible, it is indicative that this malady is caused by a virus or strains of viruses. This is further substantiated by the fact that plots treated with Thimet for the eradication of aphids in 1956-57 did not exhibit yellow-leaf, whereas non-treated aphid infested plots adjacent to such plots did exhibit the disease. Also in 1957-58, Thimet-treated plots not showing aphids at time of treatment did not exhibit yellow-leaf, but the early plantings which were heavily infested at time of treatment did show heavy yellow-leaf infestations.

Oat yields in Georgia in 1957 varied from almost nothing to over 100 bushels per acre, with test weights ranging from 17 to 35 pounds per bushel. These losses are attributed to crown rust (Puccinia coronata), victoria blight (Helminthosporium victoriae), Helminthosporium leaf spot (H. avenae), and yellow-leaf. However, on one farm it was observed that oats heavily infected with yellow-leaf and only very lightly infected with crown rust and H. avenae were reduced 50 percent in yield from 1956. Therefore, it is believed that the greater part of the damage was caused by yellow-leaf and a lesser amount by H. avenae and crown rust.

Tests are being conducted in an attempt to determine the particular virus or strains of viruses involved.

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GEORGIA EXPERIMENT STATION, EXPERIMENT, GEORGIA

EFFECTS OF STEM RUST OF DIFFERENT INFECTION INTENSITIES ON YIELD AND OTHER FACTORS IN WHEAT[1]

W. N. Garrett, M. C. Futrell, and I. M. Atkins[2]

Summary

The number of early-season stem-rust-infection centers that developed in a wheat field was found to be a critical factor in reducing yield, bushel weight, and kernel weight. Tests conducted during the 1957-58 season showed that the susceptible Bowie variety was damaged more by stem rust than was the moderately susceptible Lee variety. On plots with 24 inoculation centers per plot (7260 per acre), Bowie yielded 18.2 bushels and Lee 32.3 bushels per acre. When 8 centers (2420 per acre) were placed in each plot Bowie yielded 27.1 bushels and Lee 33.0 bushels, and with one center per plot (303 per acre) Bowie produced 27.3 and Lee 34.8 bushels per acre. Spraying with maneb produced an almost equal potential yield of 57.3 bushels for Bowie and 61.3 bushels per acre for Lee. Tests conducted in the 1956-57 season showed that the Bowie variety increased in yield from 2 to 22 bushels per acre, and the test weight increased from 42 (sample grade) to 60 (No. 1 heavy) pounds per bushel when sprayed with maneb.

Stem rust (Puccinia graminis tritici Eriks. & E. Henn.) is probably the most destructive, widely prevalent disease of wheat. Numerous studies have been made to determine the effects of stem rust and leaf rust (P. recondita Rob. ex. Desm.) on the yield of wheat. However, relatively few studies have been made to determine the damage done by stem-rust infections of different intensities. Suneson (11), Waldron (12), and Burnham (1) in the United States, Greaney et al. (7) and Peturson et al. (10) in Canada, and others (6, 9) proved that rust caused appreciable reduction in yield of wheat. Suneson (11) used isogenic lines to measure the damage caused by stem rust. Greaney et al. (7) studied the effect of stem rust on yield, chemical composition, milling and baking properties of wheat. Analysis of yield data for 1934, 1935, and 1937 showed that rust reduced yield; and 90 percent rust infection reduced the yield 34 percent and lowered the commercial grade from Manitobe No. 1 hard to "feed wheat" grade. Further analysis showed close association between stem-rust damage and protein, phosphorus, calcium, and ash contents of the grain. Ekbote and Kalamkar (4) demonstrated that time of sowing and the variety grown in an area affected stem-rust development. Hart and Hayden (8) showed that Lee wheat inoculated with race 11 of stem rust produced only 85 percent as much as the uninoculated wheat, while the same variety inoculated with race 15B yielded 60 percent as much as the control. The Lee variety gave a type-1 reaction to race 11 and a type-4 reaction to race 15B. According to Waldron (12), stem rust reduced kernel weight by shriveling. Gokhale and Patel (6), working in India, showed that a stem-rust infection covering 90 percent of the stem area reduced grain yields from 90 to 80 percent. Chester (2) worked out a method for determining rust damage. Burnham (1) estimated that the severe epidemic of race 15B on durum wheat in North Dakota in 1953 reduced yields by 45 million bushels and cut farm income by $100,000,000.

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[1]Texas Agricultural Experiment Station Technical Article No. 2999. The research work on which this report is based was conducted cooperatively by Crops Research Division, Agricultural Research Service, United States Department of Agriculture, the Graduate School and the Texas Agricultural Experiment Station of the Texas Agricultural and Mechanical College System, College Station, Texas.

[2]Respectively, Graduate Assistant, Agricultural and Mechanical College of Texas; Plant Pathologist and Agronomist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, and Texas Agricultural Experiment Station, College Station, Texas.

The purpose of the study reported was to investigate the loss in yield and other factors of wheat caused by varying the number of stem-rust-infection centers in a given area of wheat and to learn how such yield and quality could be increased by controlling stem rust with maneb.

MATERIALS AND METHODS

Bowie wheat was grown in 12 x 12-foot plots in the 1956-57 season alternately with Alamo oats in plots of the same size for isolation purposes. Bowie was used again in 1957-58, but the alternate plots were seeded to Lee wheat. Race 29 of wheat stem rust was used to inoculate the Bowie plots in both years, and race 15B was used to inoculate the Lee plots. Bowie is very susceptible to race 29 and resistant to 15B. Lee is moderately susceptible to race 15B but resistant to race 29. This difference in susceptibility made it possible to compare losses caused by stem rust on a moderately susceptible variety (Lee) with those on a very susceptible variety (Bowie).

Treatments were the same both seasons, as follows: 1) Inoculations were made at 2-foot intervals or 24 inoculations per plot, equivalent to 7260 centers per acre. 2) Inoculations were made at 4-foot intervals or 8 inoculations per plot, equivalent to 2420 centers per acre. 3) Inoculations were made at 6-foot intervals per plot equivalent to 303 centers per acre. 4) No inoculations were made. 5) No inoculations were made and plots were sprayed with maneb (6 pounds per acre in 30 gallons of water) once a week for 9 weeks. The plots were arranged in random order and each treatment was replicated four times.

Wheat plants in all plots were inoculated by a method described by Geis et al. (5). By using this method it was possible to get one or a few rust pustules per inoculation instead of the mass of pustules produced by other methods. However, because of adverse weather conditions some inoculations failed, and to fill in inoculations not established, plants were reinoculated by the hypodermic-needle method 1 month later each year. The hypodermic-needle inoculation produced large masses of pustules and caused approximately as much loss at harvest as did the centers established by the cellophane-tape method earlier when the wheat was in the seedling stage.

The date that each generation of stem rust broke and the diameter of each center were recorded in the 1956-57 season. After the identity of each infection center was lost, prevalence of rust was recorded. When 100-percent prevalence was reached, severity notes were taken until the wheat was ripe.

A rod-row sample was harvested for yield and samples were dried in an artificial dryer. Yields, bushel weights of commercial grades and 1000 kernel weights were taken. Statistical analysis was run on the yields in 1956-57 and on the yields and 1000 kernel weights of both varieties in 1957-58 by methods described by Duncan (3).

EXPERIMENTAL RESULTS

Sizes of the stem-rust centers differed in early stages of development in both years. Development of centers was rather uniform by February of 1957. The first-generation rust centers broke out about December 30 in the 1957-58 season; the centers did not remain active and were thought to be lost. However, the second-generation pustules broke on February 10. The poor development of pustules was attributed to cold and rainy weather and to bacterial action. The second-generation pustules were abnormal. The third-generation pustules broke on March 8, 1958, and by this time the rust appeared to be more normal on the plants of both Bowie and Lee varieties. This depression of rust development resulted in less damage to the wheat plants at early stages of growth and a slower spread than during the 1956-57 growing season. The identity of infection centers was lost after three rust generations in the plots that had 24 inoculations per plot and after six generations in plots having 1 and 8 inoculations per plot in the 1956-57 season.

The rate of stem-rust spread varied between growing seasons and on varieties (Tables 1 and 2). In the 1956-57 season 100 percent prevalence occurred in plots with 24 centers on February 28, in plots with 8 centers on March 22, in plots with 1 center on March 29, in plots with no inoculation on April 13, and in sprayed plots on April 27. Plants in plots with 24, 8, and 1 infection centers were dead from stem-rust infection on May 2, 1957; those on plots with no inoculations and sprayed plots had ripened normally. In the 1957-58 season 100 percent prevalence occurred in Bowie plots with 24 centers on March 8, in plots with 8 centers, 1 center, and no inoculations on April 10, and in the sprayed plots on May 10. Under the same conditions with the Lee variety, 100 percent prevalence was established in plots with 24 cen-

Table 1. Inoculation date, date that stem rust reached 100 percent prevalence, number of days between 100 percent prevalence and harvest date, and severity of stem rust at harvest, as affected by number of stem-rust centers on the Bowie variety in the 1956-57 season.

Number of Centers	Inoculation date	Date 100% prevalence established 1957	Number of days	Severity at harvest
24	12-8-56	2-28	65	100
8	12-8-56	3-22	42	100
1	12-8-56	3-29	35	100
Control	none	4-13	30	80
Maneb spray	none	4-27	6	15

Table 2. Inoculation date, date that stem rust reached 100 percent prevalence, number of days between 100 percent prevalence and harvest date, and severity of stem rust at harvest, as affected by number of stem-rust centers in the 1957-58 season.

Number of Centers	Inoculation date	Date 100% prevalence established 1957	Number of days	Severity at harvest
Bowie				
24	12-16-57	3-8	66	100 S
8	12-16-57	4-10	33	95 S
1	12-16-57	4-10	33	85 S
Control	none	4-10	33	95 S
Maneb spray	none	5-10	3	10 S
Lee				
24	12-16-57	4-27	16	40 MS
8	12-16-57	4-20	23	50 MS
1	12-16-57	5-4	9	40 MS
Control	none	5-4	9	35 MS
Maneb spray	none	*		

* Never reached 100 percent prevalence; only traces of stem rust at harvest.

ters on April 27, with 8 centers on April 20, 1 center and no inoculations on May 4. Plants of the Lee variety in sprayed plots never reached 100 percent prevalence.

Maneb spray was more effective in controlling stem rust on moderately susceptible Lee than on susceptible Bowie. Only traces of stem rust developed on sprayed Lee plants, while plants in the plots of Bowie reached 100 percent prevalence and 10 percent severity at harvest time. Plants in plots with 24 centers were dead on May 5. Plants on other Bowie and Lee plots were not killed by the disease and were ripe on May 13.

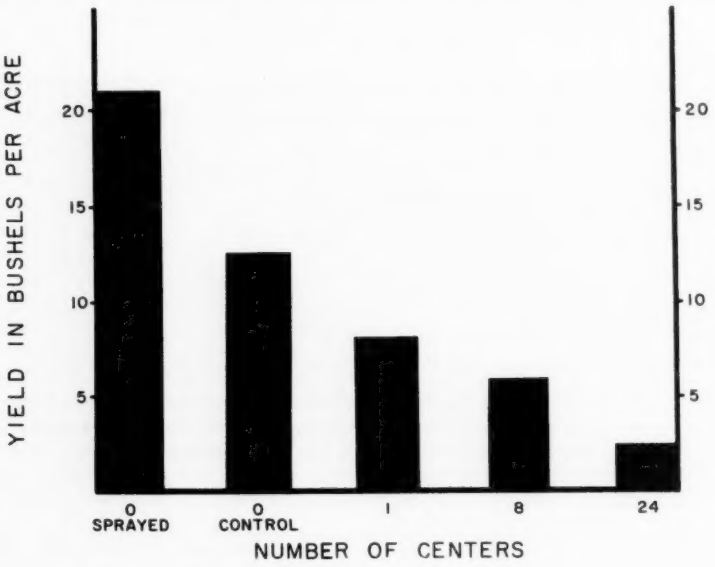

FIGURE 1. Bushels per acre of Bowie wheat produced as a result of each inoculation treatment for the 1956-57 season.

The average yields of the Bowie and the Lee variety given each treatment for both seasons are shown in Figures 1, 2, and 3. The bushel weights of Bowie for the 1956-57 season are given in Figure 4. The 1956-57 and 1957-58 data were analyzed by Duncan's Multiple Range and Multiple F Test (3). The analysis showed that there was no significant difference in replicates in either year, but that there was a highly significant difference between yields of grain of both varieties from plots of all rust treatments and the yield of grain from maneb-sprayed plots. Secondly, Duncan's Test (3) showed that the noninoculated Bowie plots yielded significantly more grain in the 1956-57 season than the plots with 1, 8, and 24 infection centers, but this was not the case in the 1957-58 season with either Bowie or Lee plots. Yields of Bowie plots with 1 infection center were not significantly different from those of plots with 8 infection centers, and yields of those with 8 infection centers were not significantly different from those with 24 infection centers. However, plots with 1 infection center yielded significantly more grain than the ones with 24 infection centers during both seasons. The Lee variety gave no significant difference in yield with 0, 1, 8, and 24 centers. The yield data presented show how effective a moderate type of resistance can be against a race of stem rust as compared with complete susceptibility.

The 1956-57 plots of Bowie with 24 infection centers yielded only 11 percent as much as the sprayed plots, while in the 1957-58 season they yielded 31 percent as much as the control plots. Environmental conditions were much more favorable for growth of the wheat plant in the 1957-58 season. Making the same comparisons for Lee grown in the 1957-58 season showed that plots with 24 centers yielded 52 percent as much as the sprayed plots.

The effect of each treatment on the weight of 1000 kernels and bushel weights was similar. One thousand kernels from the maneb-sprayed plots of both varieties in both years weighed significantly more than equal kernels from all other treatments. The 1000 kernel weights from the uninoculated plots were significantly heavier than kernels from plots with 1, 8, and

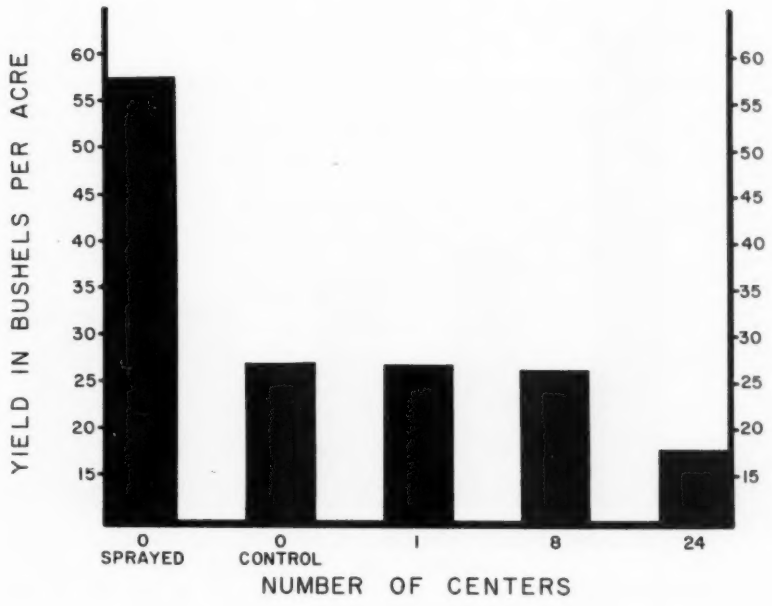

FIGURE 2. Bushels per acre of Bowie produced as a result of each treatment for the 1957-58 season.

24 centers on Bowie in both years but not with Lee. Compared with the spray plots Bowie kernels showed a 56 percent loss in weight caused by the heaviest rust inoculation (24 centers) in 1956-57, and a 53 percent loss in 1957-58; whereas, moderately susceptible Lee had only a 43 percent loss in weight of kernels from plots with 24 centers compared with sprayed plots.

Bushel weights were taken and grain from plants given each treatment was rated according to commercial grades (Figure 4). Grain produced in Bowie plots with 24 centers in the 1957-58 season graded No. 5, with 8 centers No. 5, with 1 center No. 3, with no inoculations No. 2, and with no inoculations plus maneb spray No. 1 heavy. Grain from the Lee plots with 24 centers graded No. 1 and from all other plots graded No. 1 heavy.

DISCUSSION

The results of this investigation agree with previous findings (6, 7, 8) that stem rust reduces yield, kernel weight, and bushel weight. Quantitative data on amount of infection revealed that a susceptible variety (Bowie) was damaged much more severely than a moderately susceptible variety (Lee). Likewise, the importance of seasonal environmental factors on the damage to Bowie by race 29 is demonstrated by difference in yields in the two seasons. These conclusions are in general agreement with those of Hart and Hayden (8).

For many years the common belief has existed that wheat is a too low income crop per acre to spray for the control of diseases. Data in this study point up the value of developing a fungicide that could be used for control of rust. With the current market price of wheat, spraying would be highly profitable if yields could be increased from 18 to 57 bushels per acre, as was done with the Bowie variety in this experiment. Further value of sprays was shown by the increased bushel weight of this variety. Milling and baking tests showed that maneb had no effect on this quality of wheat in this experiment.

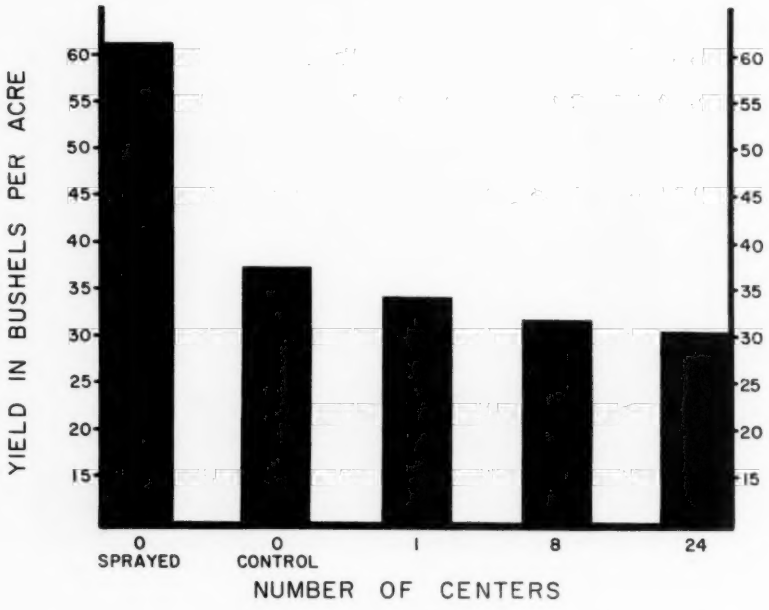

FIGURE 3. Bushels per acre of Lee produced as a result of each treatment for the 1957-58 season.

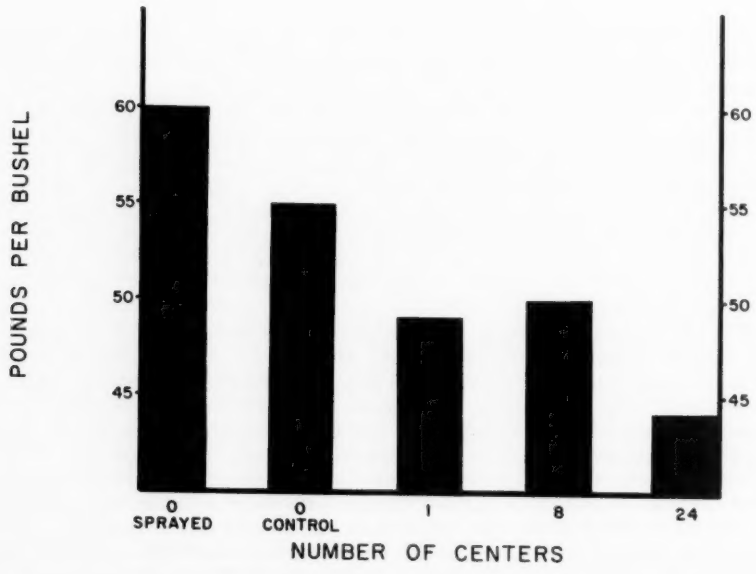

FIGURE 4. Bushel weights of Bowie produced as a result of each treatment for the 1956-57 season.

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THE TEXAS AGRICULTURAL EXPERIMENT STATION AND CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, COLLEGE STATION, TEXAS

PHYSIOLOGIC RACES OF PUCCINIA RECONDITA (P. RUBIGO-VERA F. SP. TRITICI) IN THE UNITED STATES IN 1957[1]

C. O. Johnston[2]

Summary

Twenty-four physiologic races of Puccinia recondita were isolated from 332 collections of leaf rust made in the United States in 1957. Physiologic race 15 was the most abundant and widely distributed, followed in order by races 5, 122, 54, and 105. Race 58 remained the most important race in the northeastern area and around the Great Lakes. Three comparatively new races were 130, 140, and 143.

Collections of leaf rust on wheat were made at many places in the United States and sent to Manhattan, Kansas, for physiologic race determinations. Collections representing 28 States were cultured and analyzed. They yielded 997 sub-cultures, among which 24 races were identified. Results obtained are shown in Table 1.

Race 15. Far the most abundant race in 1957, race 15 (35.8 percent of all isolates) replaced race 122, the most abundant in 1956. Race 15 was isolated from collections made in all but six States and in all of the important wheat-growing areas except the Pacific Northwest.

Race 5, representing unified race 5 was second in abundance, comprising 15.2 percent of all isolates. However, unified race 5 was fourth in importance, exceeded by unified races 6 and 13. Race 5 was collected in all but nine States and in all of the major wheat-producing areas except the Northwest. It was the most abundant race in the United States for many years prior to 1956, when it was replaced by race 122.

Race 122, one of the principal constituents of unified race 13, was third in abundance in 1957, comprising 8.4 percent of all isolates. Unified race group 13 was second in abundance, owing to the relative abundance of constituent races 54 and 122. Race 122 also was found in all wheat-growing areas except the Pacific Northwest. It was particularly abundant in the southeastern, central, and north-central areas.

Race 105, one of the principal constituents of unified race group 6, was fifth in abundance in 1957 but was not so prevalent as in 1956. Unified race group 6 was third in importance. Races 6 and 105 were the principal constituents of group 6.

Race 58 was the most important race in New York but was also abundant in Ohio, Michigan, and Virginia.

Race 11 was isolated from collections made in nine States but was important only in New York, Kansas, and Idaho. There is good evidence that it is coming into the hard red winter wheat area from Mexico. It has long been an important race on the Pacific Coast.

Race 1 was isolated only from a collection made in Kansas, and race 2 was isolated only from a collection made in Wyoming.

Races 130, 140, and 143, isolated only a few times each, are comparatively new races in the United States. Race 143 was isolated once from 1956 collections.

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[1] Contribution No. 522, serial No. 676, Department of Botany and Plant Pathology, Kansas Agricultural Experiment Station, Manhattan, Kansas, in cooperation with the Crops Research Division, Agricultural Research Service, United States Department of Agriculture.

[2] Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture.

Table 1. Physiologic races of Puccinia recondita isolated from collections of leaf rust of wheat made in the United States in 1957.

State	Number of isolates of unified races and of constituent physiologic races																										Total Number		
	1	2		3		5		6				9				10		13					17				Isolates	Races	Collections
	1	2	15	58	84	5	52	6	28	105	126	9	13	19	20	11	131	35	54	77	122	130	140	143			
Ala.			15			6		3	1	1	2	2				2		3			1				36	10	17
Ark.			1		2																1				4	3	2
Colo.			4			2	1	1		5	1	2			5					1	2				24	10	6
Fla.			20	1		10			1	3	1								1		1				38	8	12
Ga.			7	1		15		2		5	1							1	11		11				54	9	18
Idaho											1					3	1								5	3	1
Ill.			3					3		3								2	1		6				18	6	7
Iowa			41	3		6		4	3	2	2	2			2				3		2				70	11	22
Kans.	2		67	1		33		4	3	4	8	3		1	2	4		1	13	1	7		1	2	162	18	50
La.										1											3				4	2	1
Md.			2	3																					5	2	2
Mich.			5	6		3		6		6	3	1				1		1	5	1	6	1			45	13	13
Minn.			61			18		1	2	11	1	2		4					9	2	1				112	11	36
Miss.			12	1		5				1				1		1			12		10				43	8	12
Mo.			7	1		5				4	2	1				1					3				24	8	8
Nebr.			20			12		3	2	3	5			1							2				48	8	14
N. Y.				15						3						5									23	3	7
N. Car.			4	2		3				1	1				1						1				13	7	6
N. Dak.										1		1													2	2	2
Ohio				5					2			1							3		1				12	5	5
Okla.					3	1																			4	2	1
S. Car.			28	1		8													7		1				45	5	15
S. Dak.			1			8		8	1	1	4				2				1		1				27	9	7
Tenn.			3																1		4				8	3	2
Tex.			21			8		9		6		3		2	6	3			1		10	1			69	11	34
Va.			19	5	1	4	1			5									5		3	1			44	9	15
Wis.			8			3		3		7	1		1						2		6				31	8	9
Wyo.		1	8			2		5		2	1	1			2	2			2		1				27	11	7
Total	2	1	357	45	6	152	2	52	15	75	34	13	1	14	20	21	1	8	77	5	84	3	1	2	997	24	332
Percentage of total isolates	0.2	0.1	35.8	4.5	0.6	15.2	0.2	5.2	1.5	7.5	3.4	1.9	0.1	1.4	2.0	2.1	0.1	0.8	7.7	0.5	8.4	0.3	0.1	0.2			
Percentage by race groups	0.2	35.9		5.1		15.4		17.6				5.4				2.2		17.7					0.3				

CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, AND KANSAS AGRICULTURAL EXPERIMENT STATION, MANHATTAN, KANSAS

PHYSIOLOGIC RACES OF CROWN RUST OF OATS IDENTIFIED IN 1957[1]

M. D. Simons and L. J. Michel[2]

DESCRIPTIONS OF NEW RACES

Since the last compilation of previously unnumbered races of crown rust (Puccinia coronata Cda. var. avenae Fraser & Led.) was made[3], the discovery of four new races has been brought to the attention of the authors. Reactions of the 10 standard crown rust differential varieties are shown in Table 1.

Table 1. Reaction of standard differential oat varieties to four new races of crown rust.

Race Number	Reaction of indicated variety									
	Anthony	Victoria	Appler	Bond	Landhafer	Santa Fe	Ukraine	Trispernia	Bondvic	Saia
294	R	R	S	S	S	S	S	R	R	R
295	S	R	S	S	S	S	S	R	R	R
296	R	R	R	S	R	R	R	R	R	S
297	R	S	R	R	R	R	S	R	R	R

Races 294 and 295 were discovered in the United States by the authors. These races are very similar, differing only in the reactions induced in Anthony. The reactions of Anthony to these races, as well as to many other races, are somewhat variable. Consequently, the distinction between races 294 and 295 is not always clear. These races are, for practical purposes, very closely related to races 290 and 293[3]. All four of them severely parasitize the varieties Landhafer and Sante Fe, which are being widely used as sources of resistance to crown rust. Victoria, Trispernia, and Bondvic are resistant to this group of races.

Race 296 was discovered in Canada by Peturson[4], and race 297 in Sweden by Gustavsson[5]. Neither of these races is of special significance in breeding crown-rust-resistant oat varieties.

PREVALENCE AND DISTRIBUTION OF RACES IN 1957

In terms of the physiologic race picture, 1957 was a particularly eventful year for crown rust. For the past several years the number of crown-rust collections received has averaged around four or five hundred. In 1957, however, the widespread and severe occurrence of the disease and the appearance, in quantity, of new and threatening races resulted in the furnishing by cooperators of almost 1000 collections for identification. Many of these collections were made in the cooperative Uniform Oat Rust Nurseries located throughout the principal oat-producing regions of the United States. A total of 938 isolates from this material was identified (Table 2).

When the relative prevalence of the principal races in 1957 is compared with corresponding data for the last several years, two striking developments are obvious. The first concerns the prevalence of races attacking Victoria, and the second, the prevalence of races attacking Landhafer.

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[1] Cooperative investigation of the Crops Research Division, Agricultural Research Service, U. S. Department of Agriculture, and the Iowa Agricultural Experiment Station. Journal Paper No. J-3488 of the Iowa Agricultural and Home Economics Experiment Station, Ames, Iowa. Project No. 1176. The authors wish to acknowledge their indebtedness to the investigators in other countries who have generously made their data available and to those in the United States who have supplied crown-rust collections for identification.

[2] Pathologist and Agricultural Aid, respectively, Crops Research Division, Agricultural Research Service, U. S. Department of Agriculture.

[3] Simons, M. D. 1957. Physiologic races of crown rust of oats identified in 1956-57. Plant Disease Reptr. 41: 970-972.

[4] Unpublished data furnished by B. Peturson, 1957.

[5] Unpublished data furnished by Arne Gustavsson, 1957.

Table 2. Physiologic races of P. coronata avenae isolated from susceptible and from differential varieties of oats grown in North America in 1957.

State or country	Prevalence and distribution of physiologic race																								Totals	
	201	202	203	205	210	211	212	213	216	226	235	236	237	258	259	264	274	276	279	280	284	285	290	293	294	295	Isolates	Races
Ala.		1							10					1		1											13	4
Ark.		1							1																		2	2
Canada		4	1					2	4							2											14	6
Colo.		1						4	1							2							1			1	9	5
Fla.		14	6	1				10	73		3	1	1	3		25	1		2				11	4	1	2	167	16
Ga.		11	1					2	25	1						3											43	6
Ill.		6	4	1				8	5							2							1				30	7
Ind.									3							2							2	1		1	9	5
Iowa	4	7	6	2		1		6	31		1		1			16	6						7	1		2	93	14
Kans.	2	4	4				2	9	22							2	2					3		1			50	10
Ky.	3	4	1			1		1	11				1	1			2								1		26	9
La.			1					1	1																		3	3
Mexico		1							1																		2	2
Mich.		8	2					14	6															1			33	5
Minn.		4	4					2	24							8			2				2	1			44	8
Miss.		10	6					8	10							2	2									3	41	7
Mo.		2	4					6	17		1					3			2				3	1		1	40	10
Nebr.		4	3					6	10							4											27	5
N. Y.		3			1	1	3	3	10							2				1	2		1				27	10
N. Car.	1								1																		2	2
N. Dak.		4	1					1	12							6							1				26	7
Ohio	1	2						2	9							1		1	1				3	5	2	1	28	11
Okla.				1					3														2				5	2
Pa.			1					1	4						1	6											6	3
S. Car.		1						1	7							1	7						1	1			16	5
S. Dak.		2						4	23																		38	7
Tenn.	1	10	3					1								3			1				2				30	8
Tex.								13	47		1																80	8
Va.								1								5	1		1				6	3	1	1	57	11
W. Va.																												
Wis.	1	9	5	1				6	19																			
Total isolates or races	13	115	53	6	1	3	5	122	392	1	6	1	3	5	1	93	23	1	9	1	2	3	43	19	5	13	938	36
Percent of total	1.4	12	5.7	0.6	0.1	0.3	0.5	13	42	0.1	0.6	0.1	0.3	0.5	0.1	9.9	2.5	0.1	1.0	0.1	0.2	0.2	4.6	2.0	0.5	1.4		
States and countries	7	23	17	5	1	3	2	24	28	1	4	1	3	3	1	20	7	1	6	1	1	1	14	10	4	9	31	

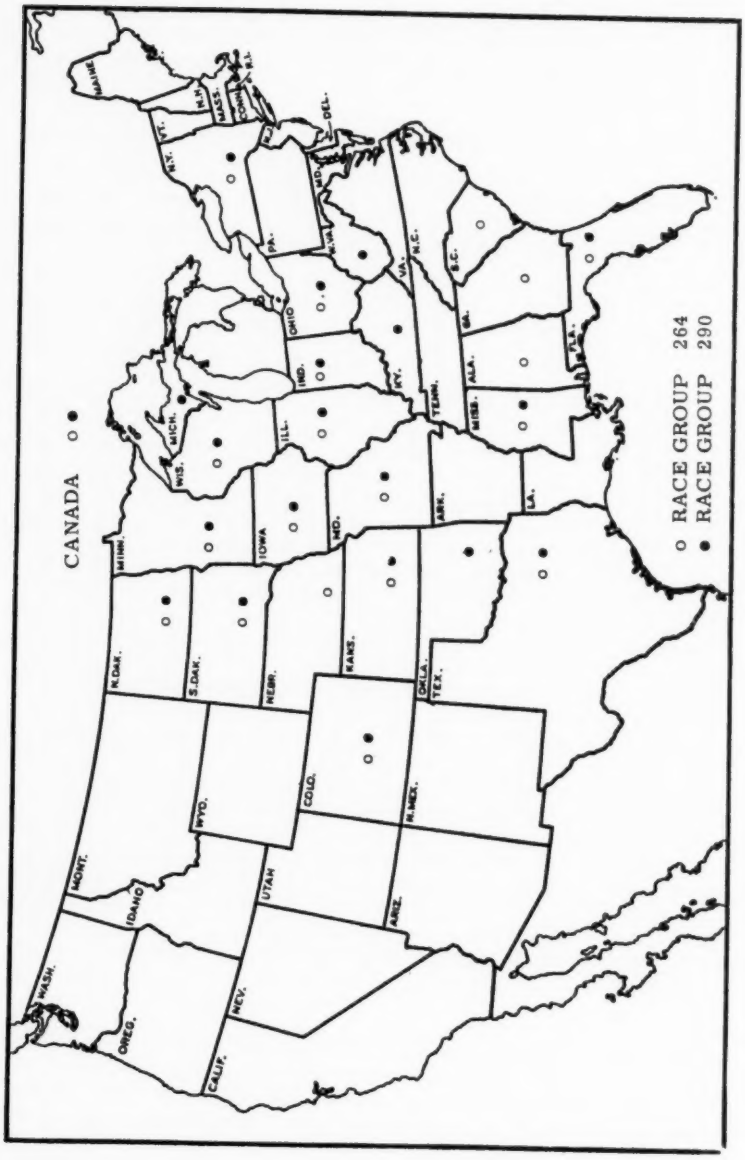

FIGURE 1. Distribution of crown rust race groups 264 and 290 in 1957.

Isolates of races attacking Victoria first appeared in appreciable numbers in 1951, when they made up about one-fifth of the isolates identified. For the next several years they showed little tendency to increase, although oat varieties of Victoria parentage were widely grown in the southern States during this period. In 1957 the eight races attacking Victoria accounted for about 70 percent of the total isolates. The increase was not divided evenly among the different races, but was concentrated in race 216. Over 40 percent of all isolates were race 216, making this the most common race identified. Race 216 is characterized by its ability to parasitize Anthony, Victoria, Appler, Bond, and Ukraine. The other differential varieties, Landhafer, Santa Fe, Trispernia, Bondvic, and Saia, are resistant. Most biotypes of this race appear to be unusually aggressive, both in the field and in the greenhouse. Victoria, which often shows some degree of resistance to other "Victoria races" in the greenhouse, ordinarily shows no resistance to biotypes of race 216 under these conditions. There is reason to think that in the field race 216 severely parasitizes certain common varieties that are moderately resistant to other races. Race 216 sometimes even appears to damage varieties such as the Landhafer derivatives, which are usually regarded as highly resistant. Race 213, another Victoria race, was the second most common race; it is distinguished from race 216 by its inability to parasitize Ukraine. From 1951 to 1956 race 202, which attacks Bond but not Victoria, was the most common race. In 1957 it dropped to third place, constituting 12 percent of all isolates. The Bond variety, which once was an excellent source of resistance to crown rust and which appears in the ancestry of the majority of the oats grown in the mid-western States, was susceptible to 20 of the 26 races identified. Ninety-eight percent of all the isolates were distributed among these 20 races.

The appearance and spread of crown rust on Landhafer and its derivatives in 1957 have already been reported[6]. Because of the interest in these races, plant breeders and pathologists made a disproportionate number of crown-rust collections from the Landhafer and other "resistant" varieties. Consequently the prevalence of the Landhafer-attacking races shown in Table 2 is not representative of their prevalence throughout the country. The Landhafer races identified in 1957 fall naturally into two groups, the 264-group (races 264 and 276) which attacks Landhafer, Santa Fe, Trispernia, and Bondvic; and the 290-group (races 290, 293, 294, and 295) which attacks Landhafer and Santa Fe, but not Trispernia or Bondvic. (The 264-group includes 263 which was not identified in 1957.) In the greenhouse Landhafer is highly susceptible to races of the 290-group. It often appears to be less than fully susceptible to race 264, suggesting that this variety and its derivatives may have some degree of resistance to race 264 in the field.

There were some instances in which the Landhafer races caused economically significant damage; however, they probably constituted only a very small percentage of the total crown rust occurring in 1957. On the other hand, it is obvious that these races were very widespread. At least one representative of these two groups of races was identified from material collected in Canada and 23 of the 29 States of the United States in which collections were made (Fig. 1). Consequently, these races even though relatively rare at present may increase rapidly as the acreage planted to Landhafer derivatives increases.

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[6] Simons, M. D., H. H. Luke, W. H. Chapman, H. C. Murphy, A. T. Wallace, and K. J. Frey. 1957. Further observations on races of crown rust attacking the oat varieties Landhafer and Santa Fe. Plant Disease Reptr. 41: 964-969.

OAT SELECTIONS RESISTANT TO CERTAIN LANDHAFER-ATTACKING RACES OF CROWN RUST[1]

H. H. Luke., W. H. Chapman, A. T. Wallace, and P. L. Pfahler[2]

Summary

The reaction to the Landhafer-attacking races of crown rust of oat selections adapted to the southern region is discussed. Certain ones were not resistant to a potentially dangerous race, and crosses that should result in resistance to all races common to this area are suggested.

The rust reactions of selections from a cross with C.I. 7172 as the resistant parent were variable under different environmental conditions. The resistance of some of these selections appeared to break down during hot weather more than did that of the C.I. 7172 parent. Moreover, C.I. 7172 and some of the above mentioned selections were evaluated for plant type and adaptability and are considered as the most promising breeding material available to southern oat breeders.

The oat variety Landhafer, introduced from Germany by H. C. Murphy in 1938 (7), has been used extensively as a crown rust resistant parent for the past decade. This variety was resistant to all known North American races of crown rust for a number of years; however, in 1953 race 263, which attacks Landhafer, was reported (3) in collections from Canada. Since that time several other races (264, 276, 290) that attack Landhafer as well as other important varieties have been found in the United States (5). Landhafer is the most important parental variety parasitized by these races; therefore, they are commonly referred to as "Landhafer races."

In 1954 race 264 was collected in Alabama and race 276 in Iowa (4). Landhafer races apparently were not well established in North America and were not identified again until 1956, when races 263 and 276 were collected in Canada (2). During the early spring of 1957 an epiphytotic of races attacking Landhafer and Santa Fe occurred in Florida. Race 264 was widespread in the Quincy area and race 290 in the Gainesville area (6). Race 290 apparently had not been collected previously in North America. These two races became widespread during the spring and summer and were reported from Florida to Canada. Race 264 was collected in North Dakota and Manitoba, and 290 was collected in Iowa and Wisconsin (6). In general, races 263 and 276 have not become as widespread as 264 and 290.

During the spring and summer of 1957 several workers screened a vast amount of material for resistance to races 264 and 290. When the resulting data were compiled, it was found that there was no satisfactory seedling resistance to race 264 among the hexaploid oats (6).

Since resistance to race 290 is available, attention has been focused on finding resistance to race 264. It is likely that selections resistant to race 264 also will be resistant to 263 and 276. Therefore, this report is primarily concerned with the selection of lines resistant to race 264 that are adapted to the southern area.

THE 1957-1958 CROWN RUST NURSERIES

Varieties and selections that held promise of being resistant to race 264 were planted in

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[1]Cooperative investigations of the Crops Research Division, Agricultural Research Service, United States Department of Agriculture, and the Florida Agricultural Experiment Station. Florida Agricultural Experiment Station Journal Series, No. 801.

[2]Pathologist, Crops Research Division, Agricultural Research Service, U. S. Department of Agriculture; Agronomist, Florida Agricultural Experiment Station; Geneticist and Assistant Agronomist, respectively, Florida Agricultural Experiment Station.

Table 1. Crown rust reaction of certain selections entered in the Florida and Puerto Rico[a] Rust Nurseries.

Group	Entry number	Selection number	Location and date of observation									
			Puerto Rico				Quincy		Gainesville			
			March 17		March 28		May 5		April 29		May 7	
			Sev.[b]	Resp.[c]	Sev.	Resp.	Sev.	Resp.	Sev.	Resp.	Sev.	Resp.
I	Irradiated Floriland											
	1	AB 73	20	MR	10	2	20	10	T	1-3	5	3
	2	AB 170	-	S	20	3	20	10	--	--	--	--
	3	AB 171	20	MR	20	3	20	10	--	--	--	--
	4	AB 172	20	MR	20	3	20	10	--	--	--	--
	5	AB 177	T	MR	10	2	20	10	10	1-3	10	1-3
	6	AB 178	T	MR	10	2	20	10	--	--	--	--
	7	AB 179	T	MR	10	2	20	1-10	--	--	--	--
	8	AB 180	T	MR	10	2	40	1-10	10	0-2	10	3
	9	AB 188	T	MR	10	2	20	1-10	--	--	--	--
	10	AB 189	T	MR	10	2	20	1-10	--	--	--	--
II	C.I. 7172 x (0-200-10[d] x Southland)											
	11	AB 201	T	MR	10	2	T	1	--	--	--	--
	12	AB 232	0	--	40	4	5	0-3	0	0	10	0-1
	13	AB 210	20	R	40	5	0	0	0	0	0	0
	14	AB 295	10	R	50	7	T	0-2	T	0-1	5	0-1
	15	AB 229	20	MR	50	5	T	0-3	0	0	10	0-3
	16	AB 213	--	S	50	7	5	0	0	0	5	0-1
	17	AB 301	--	S	60	8	5	1	0	0	0	0
	18	AB 302	--	S	60	8	0	0	0	0	0	0
	19	AB 250	40	MR	40	4	10	1	--	--	--	--
	20	AB 260	20	MR	50	5	20	0-2	--	--	--	--

[a] From processed publication by Theis and Murphy.
[b] Severity (Sev.) is the percentage of infected leaf area (Scale 0-100).
[c] Response -- R is resistant, MR moderately resistant, S susceptible; or pustule type on a 0-10 scale (0-HR, 10-VS).
[d] Minn. 0-200-10 = (Hajira-Joanette x Bond-Rainbow) x Santa Fe.

a nursery at Gainesville, Florida during the fall of 1957. Race 264 was found to be naturally present in this area during the fall and winter. Therefore, artificial inoculation with race 264 was initiated in this nursery early in February in an effort to increase this race before other races became established. The active world collection of oats (3,318 entries) was grown at Quincy, Florida and inoculated with races 203, 216, 264, and 290. The active world collection was also grown at Isabela, Puerto Rico and inoculated with race 264[3] (8). In addition, many selections from small grain breeders throughout the United States were also included in the Quincy and Isabela nurseries. Oat rusts do not occur naturally in Puerto Rico; therefore, 264 was the only race present in the Isabela nursery. This race was also predominant in the Gainesville nursery.

The Florida nurseries were exposed to an extremely cold winter followed by an exceptionally cool spring; consequently, crown rust infection was light but sufficient for readings. Observations were made during the latter part of April and the first week of May. In Puerto

[3] The authors wish to express their appreciation to H. C. Murphy (Supervisory Plant Pathologist, Crops Research Division, Agricultural Research Service, U. S. Department of Agriculture) and to T. Theis (Assistant Officer in Charge, Federal Experiment Station, Mayaguez, Puerto Rico) for data obtained from the Puerto Rico nursery that are presented in Table 1.

Rico race 264 reached epiphytotic proportions and fully susceptible entries failed to head. Readings in Puerto Rico were made during the latter part of March. Rust reactions of some of the entries resistant under both Florida and Puerto Rico conditions are presented in Table 1.

RESULTS AND DISCUSSION

The resistant selections presented in Table 1 can be separated into two rather widely different groups.

Group I (Irradiated Floriland). This group represents selections from 325 lines of irradiated Floriland. The resistance of some of these selections was first observed by W. H. Chapman at Quincy during the spring of 1957 and their origin and development has been reported (1). These Floriland selections exhibited satisfactory resistance in Puerto Rico, but all were susceptible at Quincy, where race 290 was present. Many of these selections were resistant to races 203 and 216 under greenhouse conditions; therefore, their susceptibility at Quincy was considered to be due to the presence of race 290. Although this material has exhibited adequate resistance to race 264 under widely diverse nursery conditions for two seasons, some instability has been noted. If these lines are used as resistant parental material, they should be used in combination with Trispernia or Bondvic, which possess resistance to race 290.

Group II (C.I. 7172 x [0-200-10 x Southland]) C.I. 7172 (P.I. 197278), introduced from Argentina in 1951 by the United States Department of Agriculture, was found to be resistant to race 264 at Quincy during the spring of 1957 (6). Crosses were made by W. H. Chapman in 1955, and the selections entered in the 1957-58 rust nurseries were in the F_5 generation. These selections (Table 1, entries 11-20) were resistant in Florida, but all except entries 11, 12, and 19 were rated moderately susceptible in Puerto Rico March 28. However, three (entries 12, 13, and 14) were rated resistant in Puerto Rico March 17. Possibly this breakdown of resistance was the result of high temperature in Puerto Rico during the latter part of March. The parent variety (C.I. 7172) was highly resistant at both Isabela (8) and Quincy, indicating that it is not as sensitive to high temperature breakdown as are some of its progeny. The fact that some of these selections were more susceptible at Gainesville May 7 than April 29 lends further strength to this conclusion. A recent report (6) indicated that C.I. 7172 also tends to break down late in the season or in unusually hot weather. In addition, C.I. 7172 and some of its progeny were resistant at Quincy where several races were present, indicating that some of these selections carry a good source of resistance to the most common crown rust races of the southern area.

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CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, AND THE FLORIDA AGRICULTURAL EXPERIMENT STATION

CONTROL OF COVERED KERNEL SMUT (SPHACELOTHECA SORGHI) OF GRAIN AND BLACK LOOSE SMUT (USTILAGO AVENAE) OF OATS WITH GIBBERELLIC ACID IN GREENHOUSE STUDIES[1]

Luther L. Farrar

Gibberellic acid, a metabolic product of the fungus Gibberella fujikuroi, has been tested for many and varied uses. Adequate literature reviews have been prepared by Brian et al. (5) Merck and Co. (2) and Stowe and Yamaki (30). Although the literature is voluminous on the effect of gibberellins on plant growth in general, references to its use in disease control are found very infrequently. Edwards (14) reported that Gibberella fujikuroi and G. fujikuroi var. subglutinans were antagonistic to Trichoderma viride. However growth differences were attributed to the deleterious effect of Trichoderma viride on seedlings grown from seed free of G. fujikuroi and G. fujikuroi var. subglutinans. Barton and Fine (3) found that gibberellic acid was compatible with either captan or Manzate used for the control of early blight (Alternaria solani) of tomatoes and with Agri-mycin for the control of halo blight (Pseudomonas phaseolicola) of beans. However, neither the gibberellic acid nor the fungicides modified the effect the other had when used alone. Davis and Halmos (13) using potassium gibberellate found: (a) that susceptibility of tomatoes to early blight (A. solani) was not affected, (b) that the response of tomatoes to late blight (Phytophthora infestans) was variable but there was no appreciable change in susceptibility, and (c) that pinto beans were variable in their reaction to bean rust (Uromyces phaseoli var. typica). Maramorosch (22) found that corn seedlings infected by the Mesa Central strain of maize stunt virus, China asters infected by the eastern strain of aster yellows virus, and Crimson clover (Trifolium incarnatum) by wound tumor (clover big vein) virus resumed growth after spraying three times at weekly intervals with 100 ppm gibberellic acid in water. Controls remained stunted.

The present investigations were conducted in the greenhouse during 1957 and 1958 for the control of covered smut of grain sorghum and black loose smut of oats. The basic principle for controlling smuts with gibberellic acid is discussed.

MATERIALS AND METHODS

In the initial experiment four randomized rows (3 feet long on 12-inch centers) of the grain sorghum variety pink kafir were sprayed at the 3-leaf stage, using a hand atomizer, with concentrations of 10, 100, and 1000 ppm gibberellic acid[2] in 95 percent ethyl alcohol until the upper leaf surface was thoroughly wet. Teliospores of Sphacelotheca sorghi were dusted liberally on the grain sorghum seed prior to planting 100 seed per row in soil previously fumigated with methyl bromide (2 pounds per 100 square feet). Appropriate checks were maintained. Seedlings were thinned to approximately 20 plants per row after spraying in the first experiment, when it became evident that they were too crowded for best growth. At maturity smut counts were made. The second experiment was conducted similarly, with the exception that only the 1000 ppm concentration was used, seedlings were thinned prior to spraying and at maturity, data were taken on length of internodes, height of plants from crown to the apex of the primary spike, average length of roots from the crown to the root tips, number of nodes per plant, and number of primary roots per plant. Experiments 3 and 4 were conducted with seed of the oat variety Santa Fe harvested from small grain plots at Experiment showing a high percentage of black loose smut. In both experiments, additional inoculum of black loose smut races A-14 and A-15 were used to inoculate the seed, using the partial vacuum technique. These races were collected from differential variety tests conducted during the 1956-57 growing season. Experiment 3 was conducted in a manner similar to experiment 2, except that 50 seed were planted per replication. In experiment 4, seed was treated with 2000 and 1000 ppm gibberellic acid during the partial vacuum inoculation of the seed with smut teliospores, planted to steam sterilized soil in 8-inch clay pots (10 seed per pot), 4 pots per replication. These pots were placed under two 750-watt Sylvania incandescent lights on a 20-hour day cycle controlled by a time clock. Seeds were planted February 22, 1958 and harvested April 4, 1958. Data on germination and percentage smut were recorded.

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[1] Paper No. 340. Journal Series, Georgia Experiment Station.

[2] The gibberellic acid for these studies was supplied by Eli Lilly & Company.

RESULTS

Excellent smut control was obtained at the 1000 ppm concentration in all tests except number 3, where a marked reduction was obtained (Tables 1, 2, 3, and 4). When smut-inoculated oat seed were treated with 2000 and 1000 ppm gibberellic acid by the partial vacuum technique, a very rapid and accelerated germination rate resulted (Table 5). In grain sorghum, at maturity, there was no appreciable difference in number of internodes or plant height. However, it was found that basal internodes were always shortest on plants not treated with gibberellic acid, whereas, in gibberellic-treated plants the second internode was always shortest at concentrations of 1000 ppm or greater. This is shown in Figure 1. There was no appreciable difference between number of primary roots or between average root length when comparing gibberellic-acid treated and non-treated grain sorghum. However, primary roots of non-treated plants were always larger.

Table 1. Results of treating the grain sorghum variety pink kafir in the 3-leaf stage with 10, 100, and 1000 ppm gibberellic acid spray for the control of covered kernel smut.

	Rep. I		Rep. II		Rep. III		Rep. IV		Total	
ppm	No. heads	No. smutted	No. heads	No. smutted	No. heads	No. smutted	No. heads	No. smutted	No. heads	No. smutted
1000	7	0	18	0	16	0	9	1	50	1
100	8	3	12	6	21	10	16	9	57	28
10	12	8	14	4	16	5	12	3	54	20
Check	14	5	13	4	14	9	16	7	57	25

Table 2. Results of treating the grain sorghum variety pink kafir in the 3-leaf stage with 1000 ppm gibberellic acid spray for the control of covered kernel smut.

ppm	Rep. I		Rep. II		Rep. III		Rep. IV		Total	
1000	10	0	5	0	6	0	7	0	28	0
Check	10	0	13	1	10	1	9	2	42	4

Table 3. Results of treating the oat variety Santa Fe in the 3-leaf stage with 1000 ppm gibberellic acid spray for the control of black loose smut.

	I		II		III		IV		Total
Treatment	Total heads	No. smutted	Total heads	No. smutted	Total heads	No. smutted	Total heads	No. smutted	per cent smut
1000 ppm	63	0	40	1	28	4	31	7	7.3
Control	30	17	27	17	41	3	51	5	21.6

Table 4. Results of seed treatment tests using gibberellic acid solutions of 2000 and 1000 ppm on the oat variety Santa Fe for the control of black loose smut.

	I		II		III		IV		Total
	Total plants	No. smutted	Total plants	No. smutted	Total plants	No. smutted	Total plants	No. smutted	per cent smut
2000 ppm	7	0	8	0	7	1*	5	0	3.7
1000 ppm	6	0	10	0	6	0	6	0	0.0
Control	5	1	5	1	3	0	5	1	16.6

* This oat panicle had 8 seed and only the basal oat was smutted.

Table 5. The results of gibberellic acid seed treatment tests showing the number of emerged Santa Fe seedlings per treatment per day.

Treatment	Number seedling emerging per day				Total germinated
	3 days	4 days	5 days	6 days	
2000 ppm	2	21	4	0	27
1000 ppm	18	9	1	0	28
Control	2	8	4	4	18

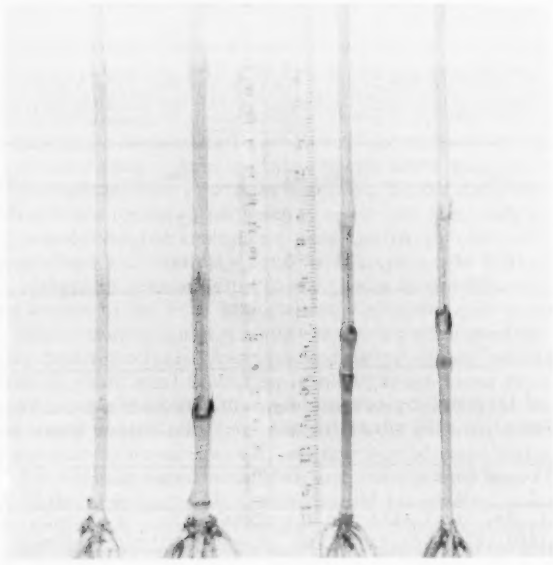

FIGURE 1. The effect of 1000 ppm gibberellic acid on sorghum stalks when sprayed in 3-leaf stage. Notice the shortest nodes of the check stalks at left are in the base.

DISCUSSION

Growth measurements from germination to maturity were not recorded since the main interest was smut control. However, in all experiments the 2000 and 1000 ppm treatments resulted in plants that were approximately twice as high as non-treated checks in 14 days. However, at maturity there were no appreciable differences in height. In experiment 1, the single smutted plant which occurred in the 1000 ppm treatment is attributed to a late germinating seed. It should be noted that plants were thinned after spraying when it became evident that they were too crowded for adequate growth. Therefore, this single smutted plant could have been from a late germinating seed. At treatment the young plant was probably shielded partially or entirely by the 3-leaf seedlings and, therefore, did not receive any, or a sufficient quantity of, spray.

In experiment 3 the replication containing the greatest percentage smut was very close to the cement and brick retaining wall. That these plants did not respond as well to the gibberellic acid treatment was attributed to the higher concentration of lime in the soil.

It should be noted in Table 4 that the single smutted oat plant contained eight seed and only the basal seed was smutted. Therefore, 3.7 percent smut for the 2000 ppm seed treatment rate does not represent a correct figure. Actually it should have received a zero smut percentage.

It is believed that the greatly accelerated germination rate in oats (Table 5) helped to reduce infection. This will be discussed later.

Although the evidence obtained in these experiments indicates that gibberellic acid controlled covered kernel smut of grain sorghum and black loose smut of oats, few generalizations can be made. However, a discussion of the possible mode or modes of control, based on pertinent literature, is deemed necessary.

Effect of Gibberellic Acid on Plants

According to Stowe and Yamaki (30) stem elongation was the most typical and striking plant response to treatment with gibberellic acid. In general, growth response was limited to younger tissues which were still growing, with wide variations even within a species, and may be influenced by external conditions and the age of the plant. Gibberellin applied to any part of the plant apparently will affect all growing parts throughout the plant. Stowe and Yamaki (30) also stated that most workers believe growth was due to cell elongation, although some workers believe that cell division must have occurred. Recent work by Greulach and Haesloop (17) with Phaseolus vulgaris and Schroeder and Spector (29) with fruit tissue indicated that cell division was stimulated by gibberellic acid. Greulach and Haesloop attributed stem elongation entirely to cell division and Schroeder and Spector found that 5 ppm gibberellic acid in the absence of indole acetic acid caused optimum stimulation of cell proliferation in excised pieces of the pericarp of Citrus medica on an agar medium.

According to Stowe and Yamaki (30), it was generally agreed that underground root growth was reduced in length, weight, and number of roots, the sole exception being Azuki bean, whose root growth was promoted. Marth, Audia, and Mitchell (23) found that gibberellic acid retarded flowering in some species but advanced it in others from one to several weeks. Brian et al. (5) found that in a compost soil containing a vigorous bacterial and fungal microflora, gibberellic activity gradually decreased to a negligible amount after 31 days. However, in sterilized soil gibberellic activity was maintained throughout the experiment.

Wittwer and Bukovac (33) found that seeds of peas (Alaska, Perfection) and beans (Michelite, Sanilac) soaked overnight in water solutions of 10, 25, 50, and 250 ppm gibberellins and planted in soil, sand, or vermiculite germinated more rapidly and emerged earlier than seedlings not so treated. At 60°F treated peas emerged 1 day earlier than checks and treated beans 3 days earlier. At 50°F differences were more marked and in many cases only treated seed emerged. Helgeson and Green (18) found that gibberellic acid stimulated germination of both primary and secondary seed of wild oats. Munekata and Kato (26) observed that gibberellin increased the elongation of barley acrospires (plumules) and rootlets at concentrations of 5 to 50 ppm. Chardon (8) treated sugarcane seedpieces in 20 and 100 ppm gibberellin and obtained growth increases over controls of 60 and 108 percent, respectively. Seedpieces dipped in 2 percent urea solution for 5 and 15 hours resulted in 40 and 85 percent growth increases, respectively, over controls.

Marth et al. (24) found that growth of barley seedlings doubled or tripled in response to 1 percent lanolin paste mixture and (23) young sugarcane plants of hybrids Co. 290, C.P. 29-116, and C.P. 44-101 responded from 93 to 286 percent over the controls with 0.5 percent lanolin paste treatment. Wittwer and Bukovac (32) obtained a pronounced growth of grass in early spring,

when growth would not otherwise occur, upon treatment with gibberellic acid. Treated plants turned lighter green but remained bright green in soils of high fertility or if fertilizer was applied. They found Poa species and Bermuda grass to be most responsive, with Zoysia not responding.

According to Brian et al. (5) and Stowe and Yamaki (30), gibberellic acid has not affected several bacteria and fungi in tests.

A Discussion of Possible Mode(s) of Smut Control

According to Fischer and Holton (16), in seedling infection types of smuts Brefeld found that oat seedlings are no longer susceptible after the coleoptile has elongated to the extent that the first leaf has extended one cm beyond its sheath. Batts (4) observed that in loose smut of wheat there is no infection down the style, but that infection occurs in the young ovary through any part of the pericarp wall and in about 10 days infection is in the testa. After 30 days infection was observed in the aleurone layer and scutellum but not in the plumule or radicle. This could explain why Vanderwalle (31) observed that in loose smut of wheat the fungus does not keep pace with the development of the host, but falls behind and finally catches up about the time the floral organs become differentiated. Ohms and Bever (27) found that Wabash and Kawvale winter wheat embryos showed no difference in percentage of infection when inoculated with races 1, 3, and 11 of Ustilago tritici. They attribute resistance in Kawvale to the apparent ability of the seedlings to outgrow the invading fungus. According to Crepin et al. (9), environment can affect one of two types of smut resistance discussed by them. In one type, the fungus is able to penetrate the external tissues of the host but fails to become established due to the incompatibility of the cell sap. In the other type, the fungus is able to penetrate to the growing point and from there develops more or less freely in the tissues, but the extent of compatibility is still so low that it is unable to sporulate (latent infection) or does so only sporadically (partial infection). Crepin et al. say that apparently environment influences only the latter type of resistance. Although reports are conflicting concerning the effect of fertilization in reducing smut infection, Fischer and Holton (16) reviewing Holton and Heald (21) state that some investigators found that nitrogen and calcium cyanamid reduced smut infection. Hiltner and Lang (20) found that infection by Tilletia tritici and T. levis was reduced to less that 1 percent on two soil types by adding 100 pounds of calcium cyanamid per acre. Infection had run from 22 to 13 percent. Heuser (19) noted that fertilization with potassium and phosphoric acid increased the percentage of bunt infection, while nitrogen reduced it by stimulating the development of the spikes. Caspar (7) found that Kainit (a form of potash) promoted development of T. tritici and T. levis, while superphosphates, basic slag, and calcium cyanamid reduced it. Rabien (28) showed that soil types and the addition of certain fertilizers influenced the percentage of infection by T. tritici. In certain cases, correlations were found to exist between high germination of spores taken from superphosphate and basic slag plots and high infection, while a correlation in the opposite direction was found between poor germination of spores taken from urea and calcium cyanamid plots and low percentage infection. In other cases, no correlation could be found and Rabien concluded that, in general, the effect of fertilizers on the infection of wheat by T. tritici is not due merely to their influence on spore germination. Feucht (15), after testing several fertilizers, concluded that calcium cyanamid was the most effective in reducing bunt infection, but it did not completely eliminate bunt. Ambastha (1), studying the relation between environment and disease resistance by monthly sowing tests of differential wheat varieties against two races of bunt, came to the conclusions that (a) genetically resistant varieties remain resistant under different meteorological conditions, viz., temperature and humidity, and (b) variations in the infection of susceptible varieties depend upon the prevailing soil and air temperature. Buchheim (6) found that even though various cereals showed an increase of infected plants in manured plots in early stages of growth, in the final result the percentage of smut was decreased, owing to the more vigorous development of the plants that allowed them to outgrow the fungus mycelium in their tissues. Such examples as 1, 9, 19, 27, 28 could explain why Melchers (25) was able to demonstrate that in susceptible varieties of grain sorghum there are numerous plants that never develop smut in the primary heads, even though mycelium of Sphacelotheca sorghi is present in their tissues.

Davis (10) and Davis and Dimond (11, 12) investigated chemotherapeutants, and chemotherapeutants as plant-growth regulators, respectively, in relation to plant resistance to a disease.

Davis (10) found that chemotherapeutic activity may be independent of fungus toxicity. Davis and Dimond (11) found two chemotherapeutants (Sodium 2-benzothiazolyl thiozlycolate and 4-chloro-3, 5 dimethylphenoxy-ethanol) increased resistance to the fusarium wilt disease of toma-

toes by altering the metabolism of the host. In 1953 they observed (12) that chemotherapeutic activity of the plant growth regulators was directly related to the interval between inoculation and treatment, the activity becoming more pronounced the longer inoculation followed treatment. They also found that plant growth regulators induced changes in the host metabolism.

According to Stowe and Yamaki (30), based on the work of several investigators, "Reminiscent of the Auxins, no convincing action of the gibberellins has yet been shown on any organism except higher plants." Brian et al. (5) reported that gibberellins had no effect on several bacteria and fungi tested.

If this is the general rule, as it apparently is, then it should be possible to treat plants infected with systemic mycelium type smuts with gibberellic acid and obtain accelerated growth only in the higher plant. The control of covered kernel smut of grain sorghum and black loose smut of oats can be explained by several of the above literature citations. For example, since both are primarily seedling type infection smuts, it is possible that an application of 1000 or 2000 ppm gibberellic acid acted as a large application of fertilizer (6, 7, 15, 19, 20, 28); or it is possible that the gibberellic acid acted as a stimulant to germination and seedling growth through cell division, causing the coleoptile to elongate to the extent that the first leaf extended 1 cm or more beyond its sheath, and resulting in its no longer being susceptible, according to Brefeld's observations (16); or it is possible that the gibberellic acid treatments caused the plants to outgrow the fungus mycelium, as Ohms and Bever (27) believed occurs in the resistance of Kawvale winter wheat to loose smut (U. tritici). It is also possible that the gibberellic acid treatment caused metabolic changes which induced resistance such as that obtained by Davis and Dimond (12). This is not likely since Barton and Fine (3) and Davis and Halmos (13) did not obtain any resistance with gibberellic acid. However, it is indicative that smut control with gibberellic acid can be effected in one of two ways or a combination of the two: 1) by seed treatment, germination is speeded up, giving less time for mycelial growth to infect, and 2) by stimulating plant growth the plant is able to outgrow the fungus.

Studies are being conducted on loose smut of wheat and barley to see if similar results can be obtained.

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GEORGIA EXPERIMENT STATION, EXPERIMENT, GEORGIA

DISEASES OF SMALL GRAINS OBSERVED IN GEORGIA DURING THE 1957-58 SEASON[1]

Luther L. Farrar and S. V. Stacy

Observations on grain diseases in Georgia were made on numerous trips and in three small grain nurseries (Experiment, Blairsville, and Calhoun). Although the State as a whole received abundant rainfall, prolonged freezes and low temperatures retarded small grain growth until March. Statewide, last winter was proclaimed the coldest in the last decade. During the 1957-58 season small grain diseases were negligible on a statewide basis. However, in certain locations severe losses occurred.

OATS

Although several diseases were observed only one caused severe damage in some counties, Helminthosporium victoriae. As early as September, stand failures were observed in Fayette County, and later stand failures were observed in Spalding, Putnam, and Merriwether Counties.

Leaf rust [crown rust] (Puccinia coronata) was observed as early as October 22, 1957, at Experiment, Georgia. However, no damage due to leaf rust was observed in fields this season.

Loose smut (Ustilago avenae) was prevalent in many oat plots at Experiment. However, only light infestations were observed in the field.

Leaf blotch (Helminthosporium avenae) was observed to a very limited extent at Blairsville, Calhoun, and Experiment.

Oat mosaic (Marmor terrestre) was observed for the first time at Calhoun, Georgia, and occurred on the variety Suregrain. It was also observed in two fields of Victorgrain in Spalding County,

Yellow-leaf (due to an aphid transmissible virus, although the specific virus, or viruses, is not known) was observed the first week in December at Experiment. Localized spots were observed in several fields in Central Georgia; however, since the severe winter kept aphid populations at a minimum, little or no spread occurred. For this reason, this disease caused much less damage this year in comparison with last year when losses were very high in certain counties[2].

WHEAT

Leaf and glume blotch (Septoria tritici and S. nodorum) were widespread. In fact, every field of wheat observed was affected by these organisms, frequently severely enough to cause reduction of kernel size.

Black point (Helminthosporium sativum) was observed in wheat fields at several locations. Bledsoe wheat from one field in Houston County averaged 16.3 percent of the kernels showing black point (Table 1). However, when this wheat was placed in moist chambers for 72 hours an average of 96.6 percent of the kernels produced H. sativum spores (Table 1). After slurry treatment with Panogen 15 no H. sativum spores could be detected after 72 hours in a moist chamber (Table 2). Figures 1 and 2 show effect of seed treatment. Notice abundant sporulation on filter paper in non-treated. The Panogen 15 was diluted with water at the rate of 5 gallons of water to one of Panogen 15 and 22 cc of this material was used to treat 8 pounds of seed. This was accomplished with a slurry treating machine after the seed had passed through two seed cleaning machines (Figures 3, 4, 5, 6). Approximately 3500 bushels of wheat will be treated in this manner by the Georgia Experiment Station.

Loose smut (Ustilago tritici) was observed in some fields in Spalding County. However, counts showed that infection was less than 0.5 percent.

Stem rust (Puccinia graminis tritici) was observed in three locations, but infection was light. At Perry, Georgia (Houston County), light infections were observed on Bledsoe and identified as race 38[3]. At Blairsville (Union County) and Haralson (Coweta County) stem rust

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[1] Contribution of Department of Plant Pathology, Georgia Experiment Station. Journal Series No. 342.

[2] Farrar, L. L. and U. R. Gore. 1957. Diseases of small grains observed in Georgia during the 1956-57 season. Plant Disease Reptr. 41: 986-987.

[3] Identified by Dr. J. J. Christensen, University of Minnesota.

was observed on Chancellor. However, infections were light and a report on the race, or races, has not been received.

Leaf rust (Puccinia rubigo-vera tritici) was observed on Chancellor wheat at Haralson and Blairsville. However, infections were late and light.

Table 1. Percent kernels showing black point in Bledsoe wheat and percent total kernels producing Helminthosporium sativum spores after 72 hours in a moist chamber.

Total kernels[a]	Number black point kernels	Percent black point kernels	Number total kernels producing spores	Percent producing spores
152	21	13.8	148	97.4
130	22	16.9	124	95.4
120	20	16.7	117	97.5
128	20	15.6	123	96.1
113	21	18.6	109	96.5
Average		16.3		96.6

[a] Five samples were taken at random from the wheat grown in Houston County prior to treatment.

Table 2. Results of treating black point kernels with Panogen 15[a].

Untreated		Treated	
Number kernels	Number producing H. sativum spores	Number kernels	Number producing H. sativum spores
100	100	100	0
100	100	100	0
100	100	100	0
100	100	100	0

[a] Black pointed kernels were hand picked from untreated seed and from a bag of certified seed 96 hours after treatment, placed in moist chambers and data taken after 72 hours.

BARLEY

Stripe (Helminthosporium gramineum), loose smut (Ustilago nuda) and scald (Rhynchosporium secalis) were observed in the nurseries at Experiment and data are presented in Table 3. Net blotch (Helminthosporium teres) [Pyrenophora teres] was very severe in a field of Calhoun planted at Experiment.

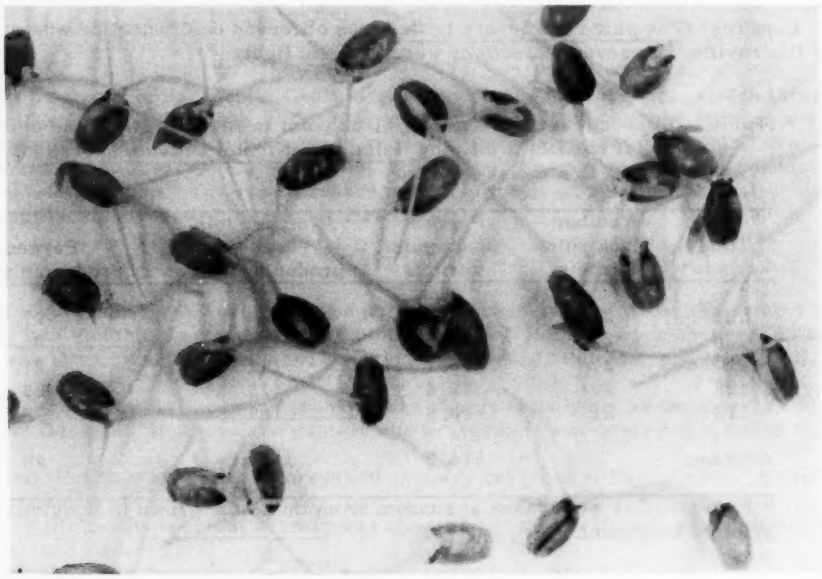

FIGURE 1. Black point affected Bledsoe wheat seed treated with Panogen 15 after 72 hours in a moist chamber.

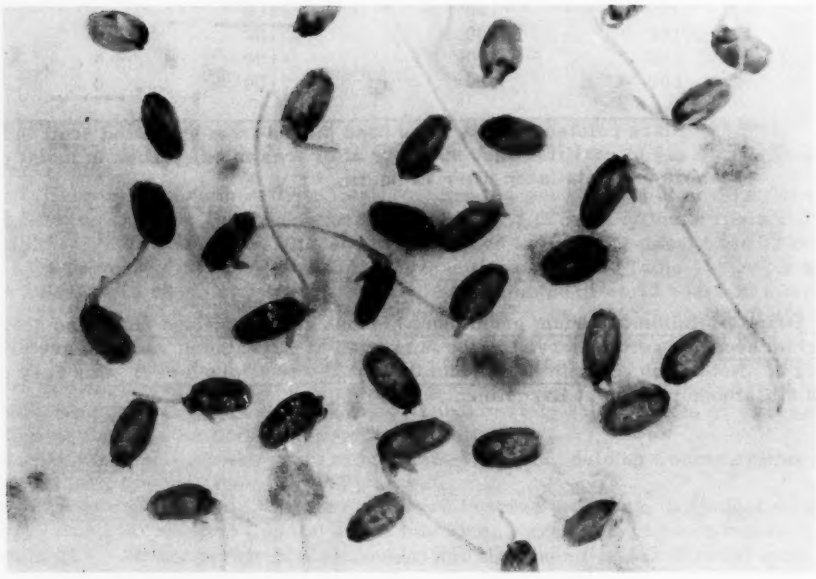

FIGURE 2. Non-treated, black point affected Bledsoe wheat seed after 72 hours in moist chamber. Notice heavy sporulation on seed and filter paper where seed were moved in counting.

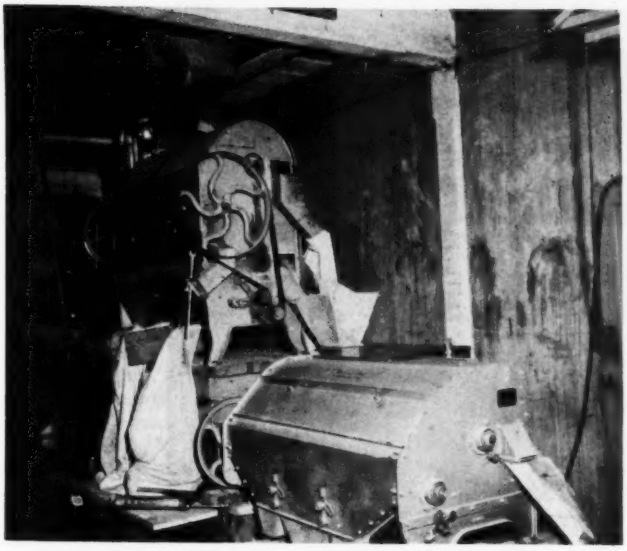

FIGURE 3. Seed are fed from clipping machine on second floor into first seed cleaner. This in turn dumps into second seed cleaner which feeds into elevator (Figure 4).

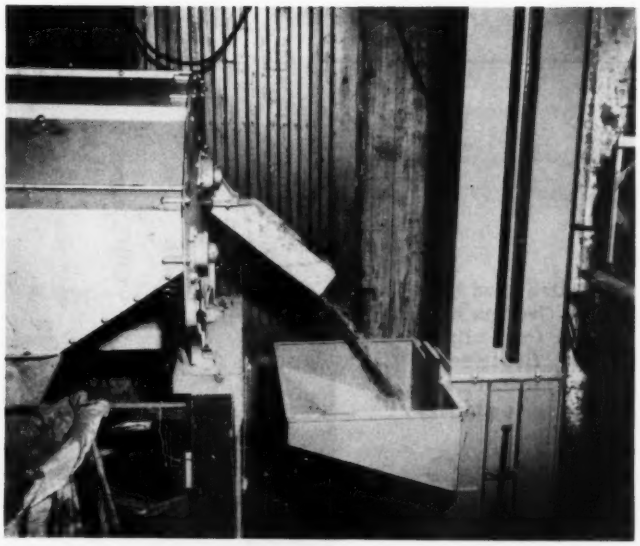

FIGURE 4. Wheat going from second seed cleaner into elevator which takes it to slurry machine.

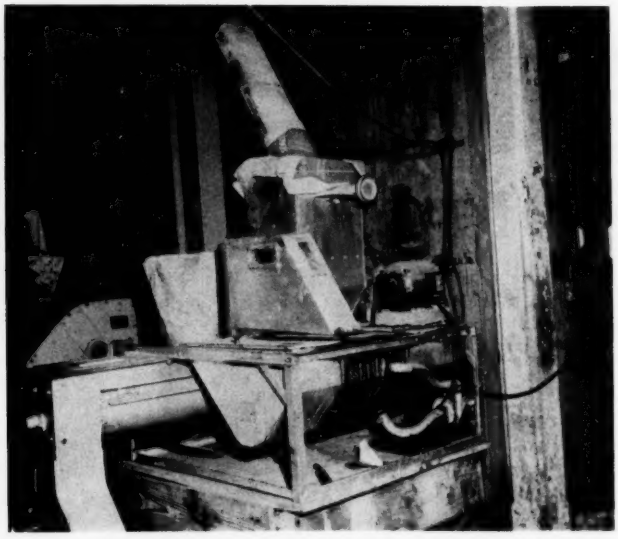

FIGURE 5. Slurry treating machine.

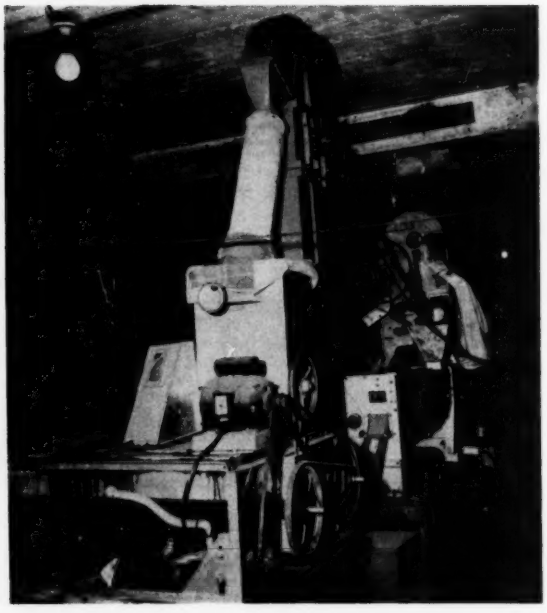

FIGURE 6. Entire seed treatment train located at Georgia Experiment Station. Hopper from clipper machine second floor, first seed cleaner, second seed cleaner, elevator to slurry machine, and slurry machine.

Table 3. Percent infection of barley varieties at Experiment by scald, smut, and stripe.

Variety	Scald	Smut	Stripe
	Total leaf area affected	Heads smutted	Plants affected
Wong	Tr	Tr	Tr
Hudson	0	0	0
Colonial	Tr	Tr	Tr
Davie	Tr	Tr	10
Cordova	Tr	15	0
Tex. 10-47-84	Tr	10	0
Tex. 10-47-136	Tr	0	Tr
Harbine	0	0	0
Rogers	0	0	15
Miss. 550-1-9	5	Tr	0
Miss. 550-2-7	10	15	Tr
Kenate	5	Tr	5
Kenbar	Tr	Tr	5
Marconee	35	5	5
Calhoun	35	Tr	0
Calhoun x Bolivar (NC. 935)	5	Tr	5
Calhoun x Bolivar (NC. 954)	10	Tr	Tr
Dayton	35	15	Tr
Wong x Jet	10	Tr	Tr
Taylor 19y443 (13 NB)	65	0	0
Taylor 19y443 (14 NB)	20	Tr	0
Georgia 8	20	0	0

RYE

Leaf rust (Puccinia rubigo-vera secalis) heavily infected Tetra Petkus rye at Blairsville and to a lesser extent at Calhoun.

GEORGIA EXPERIMENT STATION, EXPERIMENT, GEORGIA

MISCELLANEOUS NOTES ON SMALL GRAIN DISEASES IN VIRGINIA

C. W. Roane and T. M. Starling[1]

The occurrence of several unusual small grain diseases has prompted a general summary of the situation in Virginia in 1957 and 1958. No intensive survey has been made and the observations reported are from nurseries, diseased specimens sent to our laboratory, and examinations of fields of small grains along highways between Blacksburg and the various field stations. While the picture presented is rather sketchy, it does portray the trend of diseases of small grain crops for the past 2 years.

BARLEY DISEASES

In recent years scald (Rhynchosporium secalis) has become perhaps the most destructive disease of barley in Virginia. Wong (C.I. 6728)[2], the most widely grown variety, is highly susceptible to scald and the disease has been found in nearly all fields of this variety that have been examined. Hudson (C. I. 8067), a recommended, rough-awned variety with resistance to scald, is not widely grown because farmers prefer awnless types such as Colonial 2 (C.I. 8062) and Davie (C. I. 9170) and awnletted types such as Wong. Colonial 2, like Wong, is highly susceptible to scald while Davie is moderately resistant. Since Davie is not a very hardy variety and Hudson is rough-awned, it is expected that scald will continue to be prevalent in Virginia until a scald resistant, hardy variety with smooth or awnletted heads is available. In 1958 a rye variety nursery was planted adjacent to the barley nursery at Blacksburg. Considerable scald developed on the rye. No cultures were isolated for further study; consequently, it is not known whether the fungus spread to rye from barley. Scald on rye has not been reported previously from Virginia.

Leaf rust (Puccinia hordei) is generally distributed each year in Virginia but has been severe only in isolated fields. Race 4 is the predominating race. The alternate host of barley leaf rust, Ornithogalum umbellatum, is widely scattered throughout Virginia but, even in fields where barley has been grown, we have been unable to find it naturally infected with leaf rust. Bulbs of Ornithogalum have been collected and planted in isolated plots at Blacksburg. These were mulched with telium-bearing barley straw whose telia were produced in a field where race 4 predominated. Spermagonia and aecia developed on the Ornithogalum leaves in early May. Infected plants were transplanted to the greenhouse and aeciospores were transferred to susceptible varieties of barley which have been used to increase rust inoculum. Uredia appeared and spores were collected and stored. This was done both in 1957 and in 1958 but the cultures collected in 1957 were lost because the uredia were very tiny. In 1958 most of the uredia were very tiny on varieties which gave type 4 pustules with race 4. If one consults the key to races of Puccinia hordei (3), it will be noted that race 4 produces susceptible pustules on only one of the barley leaf rust differentials, namely, Egypt 4 (C.I. 6481). If, in this rust, genes for virulence are homozygous recessive and genes for avirulence are either homozygous dominant or heterozygous as is proposed by Flor for Melampsora lini (2), it is difficult to explain the reduced virulence of cultures arising from aeciospore collections. If the urediospores survive storage this summer, race determinations will be made on cultures isolated from the mixture.

Net blotch (Helminthosporium teres) and spot blotch (Cochliobolus sativus) were widely prevalent in 1957 and 1958. Net blotch was by far the most destructive of the two. In 1957 net blotch was the most destructive disease in about 20 fields examined on the routes between Blacksburg and Warsaw. By May 1 barley was severely defoliated by this disease in six of the fields examined. In 1958 net blotch was severe only in small areas of most fields where the barley was succulent. In these areas leaves were destroyed 2 or 3 weeks earlier than is normal. The occurrence of stripe in several of the fields examined indicates that there is yet much to be gained from proper barley seed treatment.

Powdery mildew (Erysiphe graminis f. sp. hordei) has not caused much damage to barley in recent years. Most of the barley acreage is sown to mildew resistant varieties such as Wong, Hudson, and Kenbar (C. I. 7574). Colonial 2 is moderately susceptible to mildew and Kentucky 1 (C.I. 6050) is highly susceptible, but the acreage of these two varieties is small.

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[1] Associate Plant Pathologist and Associate Agronomist, respectively.

[2] C. I. refers to the cereal investigation number of the United States Department of Agriculture.

Smut (Ustilago nuda) occurs in most barley in Virginia but only occasionally does it exceed 5 percent prevalence. Although Wong barley is susceptible to smut when artificially inoculated, it is seldom heavily smutted under field conditions. Calhoun (C.I. 7120), on the other hand, has virtually disappeared from production because of its extreme susceptibility to loose smut. It was difficult to maintain a supply of smut free seed suitable for certification without hot water treating the seed annually.

Heavy rains in February 1957, caused drowning of much of our barley nursery at Warsaw. Consequently, early spring growth was characterized by yellowing typical of water logging and frequently induced by Pythium root rot. It was observed, however, that Calhoun (C.I. 7120) and many of its hybrid derivatives were relatively free of this type of damage.

Sharp eye spot, a disease of cereals previously unreported from Virginia, was widespread in barley in 1958. The disease, caused by Pellicularia filamentosa (Rhizoctonia solani) is described and illustrated in Sprague's book (4) accompanied by an extensive check list. Although it is known on cereals in the western United States, it is not listed in States east of the Mississippi River except on corn. For the past 2 years, we have observed scattered strawbreaking in our barley nurseries. Since scald (Rhynchosporium secalis) has been very severe and the lesions of the sharp eye spot fungus are similar to scald lesions, the exact nature of the disease was overlooked. The scald fungus attacks the leaf sheath and not the underlying culm but the sharp eye spot fungus attacks both.

The spring of 1958 was very wet and straw breakage was observed throughout our barley nurseries at Blacksburg shortly after the heads emerged. The breakage was severe on Hudson (C.I. 8067), a variety highly resistant to scald. Counts were made to determine the relative prevalence of the disease in one of our barley variety yield tests. A random sample was taken from each of three replicates and the percentage of infected culms was determined. The disease was sporadic in the nursery but the varieties having the most breakage also had the most infected culms (Table 1).

Table 1. Prevalence of sharp eye spot on culms of barley varieties at Blacksburg, Virginia, 1958.

Variety	C.I. number[a]	Percentage of infected culms[b]
Holston	-	2.1
Colonial x Bolivia, 1341	-	4.7
Modia x Calhoun, 57-39-6	-	5.1
Colonial 2	8062	5.7
Kenbar	7574	6.1
Calhoun x Bolivia, 925	-	6.4
Kentucky 1	6050	7.5
Wong	6728	9.3
Wong x Bolivia, G38	-	13.1
Wong x Bolivia, G11	-	13.7
Calhoun x Bolivia, 938	-	15.9
Dayton	9517	16.9
Hudson	8067	20.7
Marconee	8107	20.8

[a] C.I. number is the cereal investigation accession number of the United States Department of Agriculture.

[b] Average of samples from three replicates.

Apparently, sharp eye spot was more common in the mountains of Virginia but it was also found at Orange in the Piedmont and at Warsaw in the Coastal Plains. Although we do not think it was the cause of widespread lodging, it could be found wherever lodging occurred in barley. The disease was found occasionally on wheat at Blacksburg. At present we do not consider the disease economically important but a more careful search for it will be made in the future.

OAT DISEASES

Severe reddening and yellowing of oats occurred in winter nurseries at Blacksburg, Charlotte Court House, and Warsaw in 1957. On April 24, 1957, when the Blacksburg nurseries were graded for this condition, there were very few aphids and the discoloration was attributed to the joint effects of winter injury, excessive soil moisture, and leaf spot (Helminthosporium avenae). By May 6, 1957, when most of the oats were in the boot stage and some were heading, leaf spot had become extremely severe. This condition was very conspicuous among Victoria derivatives at Charlotte Court House. Some of these varieties were almost lemon yellow but they recovered somewhat, and later were reddish-orange; their yields were very low. This is apparently the same condition which occurred in South Carolina in 1957 and was described by Byrd et al. (1). There was no recurrence of this in 1958. As a matter of fact, the winter of 1957-58 severely damaged the winter oat crop in the Piedmont and Appalachian Highlands; consequently, much of the crop was plowed down.

Powdery mildew (Erysiphe graminis f. sp. avenae), crown rust (Puccinia coronata var. avenae), stem rust (P. graminis var. avenae), the smuts (Ustilago kolleri and U. avenae) and blight (Helminthosporium victoriae) are observed each year. Of these only blight has caused severe damage to individual fields. A race of stem rust (P. g. a. 5A) was found on Saia (C.I. 4639) in 1955, 1956, and 1957 in a nursery near barberry bushes. This race is unique because it is the only race known which can attack Saia and it has not been found elsewhere (5).

WHEAT DISEASES

For the past 2 years leaf rust (Puccinia recondita) has been the most consistently damaging disease of wheat in Virginia. In 1957 leaf rust was early and heavy. Thorne, Seneca, and Vahart wheats were completely defoliated about 2 weeks prematurely in eastern Virginia, and about 1 week prematurely in western Virginia. In 1958 leaf rust appeared later and caused very little damage. In both years there was a light sprinkling of rust on Atlas 50 and 66, Taylor, Anderson and other varieties whose resistance was derived from Frontiera and Frondoso. Stem rust (P. graminis var. tritici), as usual, was prevalent in the barberry infested mountain areas but, in general, the damage from this disease was very light both in 1957 and 1958.

Leaf blotch of wheat was very severe in eastern Virginia in 1958. The disease was very active in May and destroyed all but the upper two leaves. Later the glume blotch phase became generally prevalent. Although one would expect to find Septoria tritici and S. nodorum, which have been reported from Virginia, only S. nodorum was found on the many leaves which were examined.

For several years loose smut (Ustilago tritici) has been a relatively minor disease in Virginia. Thorne, Seneca, Leap and Vahart have been the principal varieties, with Thorne predominating. All of these varieties are resistant to most races of smut. We have observed occasional smutted heads in Thorne and Vahart. Recently the seed certification agency has reported a sharp increase of loose smut in Thorne. It is apparent that a race of U. tritici new to Virginia is increasing.

Powdery mildew (Erysiphe graminis tritici) was prevalent in 1957 and caused some damage but was very light in 1958. Other diseases which were of minor importance include bunt in Carroll and Norfolk Counties, take-all (Ophiobolus graminis) at several locations, root rot in poorly drained soils of Eastern Virginia, and soil-borne mosaic at Orange. The mosaic was found at the Orange Experiment Stations; this is the first record for mosaic in Orange County.

Bunt (Tilletia foetida) has been severe only on rare occasions. Heavily bunted wheat was found in Carroll County in 1956 and near Norfolk and in Northern Virginia in 1958. Bunt in small amounts is omnipresent. The screenings collected from most flour mills will yield a considerable quantity of bunted kernels. These same screenings also contain an abundance of nematode galls (Anguina tritici).

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VIRGINIA AGRICULTURAL EXPERIMENT STATION, BLACKSBURG

A CAUSE OF PINK SEEDS IN WHEAT[1]

W. P. Campbell[2]

A small percentage of pink seeds in samples of wheat, and especially in the variety Thatcher, has been observed in recent years in Western Canada. This condition was distinct from that caused by the scab organisms (Fusarium spp.) in that there was no fungus present and the seeds had a translucent appearance.

With the advent of the combine harvester it has become common to cut the grain with a "swather" while much of it is still quite green, and to allow the grain to mature in the swath. It was thought that this practice might be related to the incidence of pink seeds in the threshed sample.

Plots of ten varieties of spring wheat were grown and harvested at four different dates, the first being while the plants were in the soft dough stage. Table 1 shows the percentage of pink seeds found in each variety cut on each date. The figures are based on counts of 1000 kernels.

Table 1. Percentage pink seeds in ten varieties of wheat harvested at 5-day intervals.

Variety	Date of harvest			
	August 8	August 13	August 18	August 23
Marquis	0	0	0	0
Redman	2.8	0	0	0
Rescue	0	0	0	0
Thatcher	2.0	4.7	0	0
Hope	0	0	0	0
Kenya Farmer	8.1	0	0	0
Lemhi	12.2	7.9	0.1	0
Lemhi X Hope-Federation	4.2	3.8	0	0
Federation	2.3	4.9	0	0
Gabo	4.0	7.1	0	0

It may be concluded from the results that harvesting a crop while it is still immature can cause seeds of wheat to take on a translucent pink appearance. It may also be noted that all varieties do not react in the same way to premature cutting.

BOTANY AND PLANT PATHOLOGY DIVISION, SCIENCE SERVICE, CANADA DEPARTMENT OF AGRICULTURE, OTTAWA, ONTARIO

[1]Contribution No. 1721 from the Botany and Plant Pathology Division, Science Service, Canada Department of Agriculture, Ottawa, Ontario.

[2]Associate Plant Pathologist, Plant Pathology Laboratory, Edmonton, Alberta.

SUSCEPTIBILITY OF LETTUCE VARIETIES AND HYBRIDS TO POWDERY MILDEW (ERYSIPHE CICHORACEARUM)

W. C. Schnathorst and Roy Bardin[1]

Summary

None of the crisp-type lettuce varieties observed, either heading (crisphead) or non-heading (leaf), were resistant to powdery mildew. Resistance was present in butter-type and cos-type varieties only.

Powdery mildew (Erysiphe cichoracearum DC. ex Mérat) was observed near Salinas, California, in 1940 on the wild-commercial lettuce hybrid (Lactuca serriola L. x L. sativa L.), 41858, by Pryor (2). Powdery mildew was again observed in 1951, and was widespread in commercial lettuce plantings in the Salinas Valley (4), which indicated that a new strain of the fungus was involved. This disease has occurred each year since 1951, and in some instances has caused serious damage. Sources of resistance to this new strain of mildew have been indicated in the literature (1), but no report has been published on the comparative susceptibility of some of the major lettuce varieties grown in the United States. This paper presents observations made on the susceptibility of various varieties of lettuce and lettuce hybrids.

Tests for susceptibility of lettuce varieties to powdery mildew give results in the laboratory that often conflict with field observations (3). Varieties apparently not susceptible in the field become readily infected as potted plants in the greenhouse or in detached-leaf culture. The degree of susceptibility in lettuce varieties was determined in a variety trial in the Salinas Valley[2] in 1954. Infection in this trial was heavy and uniform over the whole planting. Readings were made when the plants were nearing market maturity. At least 10 plants of a given variety, or cross, were examined for mildew.

The comparative susceptibility to powdery mildew of the lettuce varieties and crosses observed is presented in Table 1. None of the crisp-type varieties, either heading (crisphead) or non-heading (leaf), were resistant to powdery mildew. Resistance[3] was found in four cases: Arctic King and Big Boston, both butterhead types; Salad Bowl, a non-heading butter type; and Bath Cos, a red-leaved cos-type variety. Readings in a similar variety trial in 1955 verified the above observations.

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[1] Respectively, Plant Pathologist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, and Plant Pathologist, Monterey County Department of Agriculture, Salinas, California.

[2] Thanks are due the Ferry-Morse Seed Co., Salinas, California, for the use of their variety trials to make these readings.

[3] Resistance means no mildew colonies were observed on the plants.

Table 1. Comparative susceptibility of various varieties and crosses of lettuce to powdery mildew in the Salinas Valley, of California, in 1954.

Variety or hybrid	Varietal type	No. lines observed	Mildew reaction[a]
Alaska	Crisphead	1	+
All Year Round	Butterhead	1	+
Arctic King	do	1	-
Bath Cos	Cos	1	-
Bibb	Butterhead	1	+
Big Boston	do	1	-
Blonde Long Standing	Cos	2	+
Cornell 456	Crisphead	3	+
Denver Market	do	1	+
Elbeck Special	Non-heading butter type	1	+
Grand Rapids	Leaf (crisp)	1	+
Great Lakes	Crisphead	73	+
Great Lakes, A-36	do	2	+
Great Lakes, Lake Superior	do	1	+
Great Lakes x Continuity	Crisphead x Butterhead	3	+
Great Lakes x Imperial, E-4	Crisphead	13	+
Great Lakes x White Boston	Crisphead x Butterhead	3	+
Hanson	Crisphead	1	+
Iceberg	do	1	+
Imperial 44	do	2	+
Imperial 101	do	3	+
Imperial 10hK	do	1	+
Imperial 152	do	1	+
Imperial 615	do	4	+
Imperial 749	do	1	+
Imperial 847	Crisphead	1	+
Imperial, Triumph	do	1	+
Imperial E-4 x Iceberg	do	1	+
Imperial 615 x Great Lakes	do	57	+
Imperial 615 x Imperial E-4	do	5	+
Jade	do	1	+
K-1	do	1	+
K-1N	do	1	+
Lavagnino Special	do	3	+
MC 56	do	1	+
New York 515	do	1	+
No. 19	do	1	+
Oak Leaf	Non-heading butter type	1	+
Pennlake	Crisphead	2	+
Prize Head	Leaf (crisp)	1	+
Progress	Crisphead	1	+
Red Leaf	Cos	1	+
Salad Bowl	Non-heading butter type	1	-
USDA 3186	Crisphead	1	+
USDA 4164	do	1	+
White Boston	Butterhead	4	+

[a] + designates mildew present, - designates no mildew observed.

DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA, DAVIS, AND MONTEREY COUNTY DEPARTMENT OF AGRICULTURE, SALINAS

GROUNDWORK FOR ELUCIDATION OF THE MECHANISMS OF VARIATION OF PATHOGENICITY OF THE FUNGUS CAUSING ANTHRACNOSE OF CUCURBITS[1]

S. K. Dutta[2]

The fungus Colletotrichum lagenarium (Pass.) Ell. & Halst. is a widely recognized pathogen that causes anthracnose in cucurbits. It can attack any above-ground parts of host plants of the species Citrullus vulgaris, Cucumis melo, C. sativus, Cucurbita maxima, C. pepo and C. moschata. According to the "nutrition-inhibition hypothesis of pathogenicity," proposed by Garber (1), the avirulence is due either to the unavailability of one or more nutrilites in the host environment required by the parasite for proliferation or metabolism, or to effective inhibition of the parasite at the site of infected host.

This fungus provides opportunities to study nutritional aspects of this host-parasite relationship. The fungus is comparatively easy to culture, and its wide host range covers more than six different species of cucurbits. The present study was undertaken to determine effects of different nutritional deficiencies on the pathogenicity of induced mutants as compared with their wild type.

Cultures of different isolates were obtained from several U. S. agricultural experiment stations. Single spore isolations were made from each. Virulence of these isolates was tested against several commercial varieties and plant introduction numbers of watermelon, muskmelon, cucumber, squash, and pumpkin. On that basis, and by comparing minimal requirements, a standard virulent type of the pathogen was selected. This type was race 2, from N. N. Winstead, Department of Plant Pathology, North Carolina State College. The watermelon varieties Black Diamond and Charleston Gray, cucumber varieties Model and Long Marketer, and muskmelon varieties Delaware 7A and Hales Best 36 were found to be six suitable different varieties for comparing different natural races and artificial mutants. None of the plant introduction lines or commercial varieties of squash and pumpkin were found suitable as differential varieties.

Artificial biochemical mutants induced were obtained by treating conidia from the selected standard virulent strain with ultraviolet light. A technic to induce nutritional mutants and to separate and isolate them from the non-mutant population was developed, using the principle developed by Woodward et al. (2).

Mutants requiring the amino acids isoleucine, serine, lysine, leucine, and histidine appeared to be consistently nonpathogenic. Those requiring the vitamins pyridoxine and choline were moderately pathogenic to Black Diamond and Model varieties. The mutant requiring the vitamin inositol was found to be only slightly pathogenic to most of the varieties. One purine-requiring mutant was found to be avirulent to all the varieties.

Results of these preliminary investigations confirm the effects of certain induced nutritional deficiencies on pathogenicity. It is also evident that the pathogenic fungus C. lagenarium is an excellent organism for future investigations related to the host-parasite metabolic relationships.

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DEPARTMENT OF HORTICULTURE, KANSAS AGRICULTURAL EXPERIMENT STATION, MANHATTAN

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[1] Contribution No. 276, Department of Horticulture, Kansas Agricultural Experiment Station, Manhattan. These and other data are to be included in the author's Ph. D. dissertation.

[2] Graduate Research Assistant, Kansas Agricultural Experiment Station.

The author is indebted to Dr. E. G. Heyne and Dr. V. W. Woodward, Agronomy, Dr. E. D. Hansing, Plant Pathology, and Professor C. V. Hall, Horticulture, for assistance in this research.

INFECTION OF DANISH SEEDS BY RHIZOCTONIA SOLANI KUEHN

Paul Neergaard[1]

Summary

Seed infection by Rhizoctonia solani has been found consistently in certain species, whereas never or seldom in other species. The fungus was found in 68 out of a total of 1045 Danish seed lots of various crucifers (Table 1) and furthermore encountered in seeds of Cichorium intybus (1 of 9 lots), Dianthus caryophyllus (4 of 12 lots), Impatiens balsamina (4 of 12 lots), Lactuca sativa (1 of 170 lots), Raphanus sativus (1 of 99 lots), Spinacia oleracea (13 of 203 lots), Trifolium pratensis (2 of 4 lots), Viola tricolor (only one lot investigated) and Zinnia elegans (18 of 179 lots). None of the samples of the following seed categories were infected: grasses (1257 samples), beet (533 samples), oat (258 samples), white clover (176 samples), carrot (147 samples) and flax (159 samples).

INTRODUCTION

During the routine testing of seed-borne diseases at the Danish Government Plant Protection Service the mycelial stage of Corticium solani (Prill. & Del.) Bourd. & Galz. has been encountered in a considerable number of seed lots of different species.

Baker (1) published a comprehensive report on seed transmission of this fungus in chilli pepper, tomato, egg plant and Zinnia elegans in California, and reviewed the literature of earlier records on such transmission in bean, pea, hairy vetch, ground nut and subterranean clover in the United States of America and in spinach and Japanese hop in Denmark, to which may be added cotton in Egypt and India (3, 15). Since then additional examples of hosts on which Rhizoctonia solani is seed-borne have been reported: Agrostis tenuis (4), Dianthus caryophyllus (2), Callistephus chinensis (14), Matthiola incana (de Tempe in 14), Lolium sp. (Mary Noble in 14), and soybean (5), besides some of the hosts dealt with here and briefly reported earlier by the author (6 - 12).

Because of the predominantly soil-inhabiting nature of Rhizoctonia solani the importance of seed transmission of this pathogen has been overlooked, neglected or even denied for a long time. Baker (1) clearly points out that seed transmission of this fungus is significant because: (a) it introduces strains of the fungus to new areas or fields; (b) it contaminates disinfested soil, causing severe seedling loss; (c) it assures the continued association of a virulent strain of the fungus with the appropriate host.

MATERIALS AND METHODS

The 1045 seed lots of crucifers referred to in this paper are identical with the seed lots examined for Sclerotinia sclerotiorum, on which the writer recently reported in this Journal (13). At least 1000 seeds of each of these samples were placed in Petri dishes on filter paper moistened in a 0.2 percent solution of 2,4-dichlorophenoxyacetate in order to prevent germination of the seed. The seed were placed at room temperature and examined 5 and 10 days after sowing.

Furthermore a number of other seed species have been examined. One hundred to two hundred seeds of each sample were sown in Petri dishes on blotter paper moistened with water (about half of the zinnia samples in a 0.2 percent solution of 2,4-dichlorophenoxyacetate as done for the crucifers). The seeds were placed at room temperature and examined after a week.

[1] Senior Plant Pathologist, Government Plant Protection Service, Copenhagen, Denmark.

Table 1. Seed infection by Rhizoctonia solani in Danish seed lots of Brassica oleracea, Brassica napus var. napobrassica and Brassica campestris, 1953-1958. In parentheses, total numbers of seed lots investigated.

Variety	1953-1954	1954-1955	1955-1956	1956-1957	1957-1958	Total 1953-1958
Cabbage	1 (98)	3 (98)	(91)	22 (81)	6 (73)	32 (441)
Red cabbage	2 (32)	(24)	(19)	3 (15)	(17)	5 (107)
Savoy cabbage	(18)	(25)	(25)	(31)	(28)	0 (127)
Spring cabbage	2 (18)	(8)	(8)	(11)	2 (15)	4 (60)
Brussels sprouts	(10)	(14)	(9)	(6)	2 (20)	2 (59)
Cauliflower	9 (97)	2 (74)	1 (75)	4 (77)	2 (64)	18 (387)
Kale	1 (14)	(3)	(9)	1 (15)	1 (11)	3 (42)
Swede	1 (47)	1 (40)	(31)	(28)	1 (24)	3 (170)
Turnip	(29)	(10)	(6)	(16)	(13)	0 (74)
Other Brassica species	(4)	(48)	(11)	(14)	1 (11)	1 (88)
Total						68 (1045)

RESULTS

Rhizoctonia solani was found consistently in certain seed species whereas never in other species although a considerable number of samples of these species have been investigated through a number of years. From infected seeds the fungus spreads readily on the filter paper on which the seeds were sown, forming a conspicuous, brownish mycelial cobweb.

In accordance with what Baker (1) found in California, zinnia seeds are rather frequently infected. In the 5-year period 1953-58 the fungus was encountered in 18 of 179 samples of Zinnia elegans or in about 10 percent of the seed lots (most of which have been produced in Italy).

The fungus is also rather frequent in seeds of various crucifers, as it appears from Table 1 (compare this table with Table 1 in (13) for occurrence of Sclerotinia sclerotiorum in the same seed lots). In two of the 68 seed lots infected by Rhizoctonia the seed was concurrently infected by Sclerotinia.

Furthermore the fungus was found in one seed lot of Viola tricolor (only one lot investigated), in lettuce (1 of 170 lots), and in radish (1 of 99 seed lots).

Finally earlier investigations by the writer revealed the fungus in 13 of 203 seed lots of spinach (6, 8), in 1 of 9 seed lots of chicory root (8), in 1 of 59 seed lots of Dianthus caryophyllus (8), in 4 of 12 seed lots of Impatiens balsamina (7), and in 2 of 4 seed lots of red clover (8).

During the seed testing carried out in the 5-year period 1953-58 Rhizoctonia solani was not found, for example, in 533 seed samples of various varieties of beet, in 258 oat samples, 176 white clover samples, 147 carrot samples and 159 flax samples. Finally the fungus was found in none of 1257 grass seed samples, that is, 318 samples of Dactylis glomerata, 168 Festuca pratensis, 234 Festuca rubra, 69 Lolium muliflorum, 284 Lolium perenne, 124 Poa pratensis and 60 Poa trivialis.

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GOVERNMENT PLANT PROTECTION SERVICE, COPENHAGEN-HELLERUP, DENMARK

USE OF SLICES OF CARROT AND OTHER FLESHY ROOTS TO DETECT CROWN GALL BACTERIA IN SOIL

Peter A. Ark[1] and Milton N. Schroth[2]

Abstract

Crown gall bacterium, Agrobacterium tumefaciens, forms good galls on slices of carrots, rutabagas, turnips and table beets. Carrot root slices are found to be the best in detecting living organisms of crown gall in soil and galls can be seen on carrot slices after 5 or 7 days.

INTRODUCTION

It is of common knowledge that the crown gall pathogen, Agrobacterium tumefaciens, inhabits many types of soils that are used for growing a variety of crops susceptible to crown gall. At the present time there is no method known for ascertaining the presence of the pathogen in a given soil prior to planting crops that are likely to be attacked, such as almonds, peaches, cherries, etc. Losses that follow the development of crown gall on susceptible rootstocks may be quite high, not to mention the fact that the population increase of the pathogen in soils where susceptible species have been grown is so rapid and great that it may even force the abandonment of such land for those particular crops for a long time. Therefore, if it were possible to find some plants that could serve as indicators of the degree of crown gall infestation in a given soil, a crop failure could be eliminated and a susceptible crop could be grown. Crown gall is known to occur in nature on sugar and table beets and has been shown to develop on slices of carrots (Figs. 1, 2).

MATERIALS AND METHODS

Young fresh roots of carrots (Chantenay), table beets, rutabagas and turnips, purchased at a local market, were thoroughly cleaned, washed, and air dried at room temperature. Large size Petri dishes lined with wet sterilized filter paper were used for carrots, and sterilized pyrex moist chambers lined with moist paper towels for the other roots. Slices one-half inch and thicker were used. Six different isolants of Agrobacterium tumefaciens from six different hosts were grown on potato-dextrose-peptone agar slants for 24 hours at 28° C and pooled before being applied as inoculum to the experimental root slices. Inoculation was performed in two ways: 1) by swabbing upper cut surfaces of the slices with a cotton swab saturated with a heavy suspension of the pathogen, or 2) by dipping the slice into the bacterial suspension, then placing it in the moist chamber or Petri dish. In another series of experiments sterilized and non-sterilized greenhouse soils, as well as sterilized sand, were contaminated with A. tumefaciens and carrot slices were buried in these soil samples for a certain length of time at various temperatures. A number of tests were also carried out on the recovery of the pathogen from soil solutions by the use of carrot slices.

RESULTS AND CONCLUSIONS

Agrobacterium tumefaciens, whether from the slant or from soil extracts, can induce visible tumefactions on slices of carrots, table beets, rutabagas and turnips within seven or more days (Figs. 1, 2). Carrot slices appear to be preferable to the others since the galls seem to develop on carrot somewhat faster and attain larger proportions with time (Fig. 3) as compared with other roots tested. Apparently the galls develop with equal ease as a result of inoculation by either swab or immersion. Carrot slices left in contaminated sand or soil also developed galls in about a week. It should be mentioned that soils which may contain rot-producing organisms may present some difficulty.

The results of a large number of replicated experiments presented here point definitely to a possible successful use of carrot slices in detecting crown gall bacteria in contaminated soils and, when perfected, this method might be used for checking the efficiency of soil fumi-

[1]Professor of Plant Pathology, University of California, Berkeley 4, California.
[2]Graduate student, Department of Plant Pathology, University of California, Berkeley 4, California.

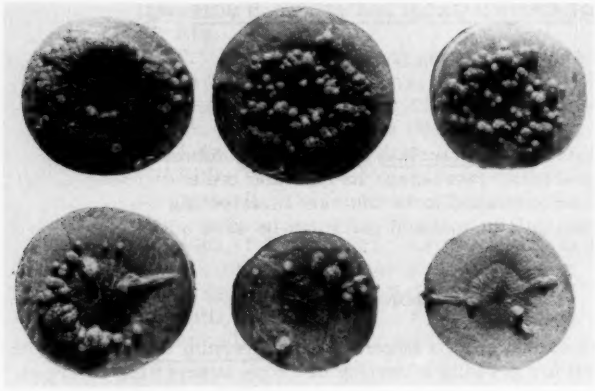

FIGURE 1. Carrot slices (Chantenay var.) showing crown galls resulting from swabbing with serial dilutions of Agrobacterium tumefaciens (heavy to light), 2 weeks after incubation at room temperature.

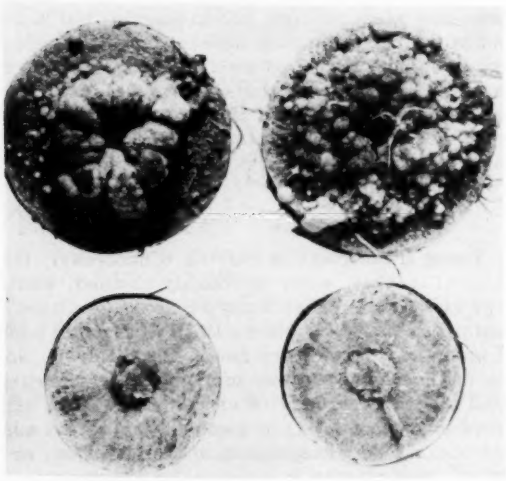

FIGURE 2. Crown galls produced by Agrobacterium tumefaciens on carrot slices (upper row), 2 weeks after inoculation by swabbing and incubation at room temperature. Lower row was swabbed with water to serve as check.

FIGURE 3. Crown galls produced on root slices of left to right, a) table beet, b) rutabaga, c) turnip, 2 weeks after inoculation and incubation at 28° C.

gation or for determining the suitability of a given piece of land for growing plants susceptible to crown gall.

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DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA, BERKELEY 4, CALIFORNIA

EFFECT OF SPRAY BLASTS UPON THE SPREAD OF A PATHOGENIC FUNGUS

Paul E. Waggoner

If a cell of a pathogen is not killed by a pesticide spray, it may be carried to a new host by the blast and find itself in a favorably moist environment. Thus spraying might increase the amount of disease in a crop. Dimock (1, 2) has shown that fungus spores suspended in pest sprays can be used to inoculate plants and has emphasized the potential role of sprays of low fungicidal value in the dispersal of viable spores. In our experiments we have tested whether or not sprays of low fungicidal activity actually can cause increased amounts of potato late blight in the field. This disease was chosen because it represents the downy mildews, diseases of economic importance that are dependent upon moisture and that occur in crops that are sprayed routinely.

Green Mountain potatoes (Solanum tuberosum L.) were planted at 1-foot intervals in rows in an open field and in a tent of cheesecloth; this provided diverse environments. In droughty 1953, the plants at the center of two sets of three rows in tent and field were inoculated with Phytophthora infestans (Mont.) d By. Seven days later the late blight lesions in which the fungus was sporulating were counted; they were confined to the center 3 feet of each row (Table 1). Later on this sunny morning, one set of three rows in both tent and field were sprayed with a 0.12 percent aqueous suspension of DDT (1;1;1-trichloro-2,2-bis(P-chlorophenyl)ethane). The spray was applied at a pressure of 200 pounds/square inch by a jet that was 1 foot wide at a distance of 3 feet from the nozzle. The jet, which was first directed through the infected plants and toward the healthy plants in the same row, was moved along the row to the end. Finally, the operation was repeated, but in the opposite direction. Later, the potatoes under the tent were sprinkled with water for 7 hours, thus increasing the difference between the field and the moist environment of the tent. The lesions evident 8 days later were counted. The mean number of lesions per 3 feet of row for three rows and two directions from the source were increased due to the spray by 50-100 percent at five out of six distances (Table 1).

Table 1. 1953.

Time	Distance from source (feet)	Late blight lesions per 3 feet of row			
		Tent		Open	
		Unsprayed	Sprayed	Unsprayed	Sprayed
Date of spray, mean of 3	0	39	27	32	32
8 days later, mean of 6	3	6	12	7	6
	6	3	6	2	3
	9	2	3	1	2

The 1956 experiment differed from the one in 1953 chiefly in that more lesions were present, spraying was done on a cloudy day, and sprinkling was omitted. Plants at the center of eight pairs of rows in both the field and tent were inoculated. The consequent source or primary lesions were counted (Table 2). Four pairs of rows in both locations were sprayed with a 0.14 percent aqueous suspension of Dilan (1 part 2 nitro-1, 1-bis(P-chlorophenyl)butane). The spray jet was directed as before. Rain fell in the afternoon. The lesions in the rows, excluding the inoculated hills, were counted 1 week later. No important change in the mean number of lesions per row was associated with spraying in the humid climate of 1956 (Table 2).

These experiments in four different environments demonstrate that no disastrous increase in the spread of a moisture-dependent fungus is produced by spray jets, especially in the important case of a wet year such as 1956. Hence, spraying with a fungicide of even modest toxicity should be useful and spraying with a non-fungicidal pesticide should never be disastrous.

Large amounts of rain and dew fall on a field and many gusts of wind pass through it; the contribution of the spray blast to water and turbulence is puny by comparison. Thus, the failure of spray blasts to speed spread significantly is not surprising.

Table 2. 1956.

Time	Late blight lesions per row			
	Tent		Open	
	Unsprayed	Sprayed	Unsprayed	Sprayed
Date of spray, mean of 6	30	46	29	37
7 days later, mean of 6	176	168	108	140

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DEPARTMENT OF SOILS AND CLIMATOLOGY, THE CONNECTICUT AGRICULTURAL EXPERIMENT STATION, NEW HAVEN, CONNECTICUT

EVALUATION OF SEVERAL NEMATOCIDES FOR CONTROL OF STING NEMATODES ON LIMA BEANS[1]

Arnold E. Steele and J. M. Good[2]

Summary

Sting nematode populations in plots treated with soil fumigants which contained a mixture of dichloropropenes and ethylene dibromide or dibromochloropropane as active ingredients were reduced as compared with those in untreated control plots, and remained low for at least 92 days after treatment. Populations in plots treated with fumigants containing ethylene dibromide or dibromochloropropane were still low 126 days after treatment, whereas populations in plots treated with fumigants not containing bromine were not significantly different from those in untreated control plots. Yields of Henderson Bush lima beans were higher from plots treated with 41 percent EDB or with DCP-EDB mixture than from plots treated with DD mixture or DCP.

In 1942 Steiner (13) described, as the type species of a new genus, a sting nematode, Belonolaimus gracilis, which was found attacking the roots of slash and long-leaf pine seedlings in various forest nurseries in Florida. Since that time, nematodes of this genus have been found in enough locations in Florida and in various places in Virginia, Georgia, North Carolina, South Carolina and Louisiana to indicate that they are widely distributed throughout the southwestern States. Where they occur in large numbers, sting nematodes have produced considerable damage on a number of different kinds of crops plants (1, 6, 7, 9, 10, 11). Christie (2, 3), stated that sting nematodes have caused greater financial loss to vegetable and strawberry growers in Florida than any other kind of nematode, including root-knot nematode.

Perry (12) reported that sting nematodes in Florida soils could easily be controlled by soil fumigation with ethylene dibromide (EDB) and other unspecified nematocides, and that populations were not re-established rapidly after fumigation. Holdeman (8) reported that plots treated with DD mixture[3], at the rate of 1.5 pints per 160 feet of row, and planted to soybeans were reinfested 2 months after application of the nematocide. Similar plots treated with 1/4 pint of 83 percent EDB[3] or 1/16 pint of DBCP[3] per 160 feet of row were not reinfested until 4 months after treatment. Good and Parham (5) treated plots with 9.5 gallons of 41 percent EDB, 0.9 gallon of DBCP, 8.0 gallons of DCP-EDB[3] mixture, or 9.5 gallons of DD mixture per acre, applied to rows 38 inches apart. Seed cotton yields on the plots averaged 1606, 1565, 1026 and 884 pounds per acre, respectively, as compared with only 203 pounds per acre on untreated plots. Ten weeks after planting, sting nematode populations were significantly lower in plots treated with EDB or DBCP than in unfumigated plots, but not in the plots treated with the other materials. At harvest time, only the plots treated with DBCP had significantly lower populations of sting nematodes than the untreated plots.

The above results strongly suggest that fumigants containing EDB and DBCP are more effective for control of sting nematodes than are materials having DCP as the principal active ingredient, since populations of this nematode remain low for a longer period after fumigation. The work reported in this paper was undertaken to determine whether differences in control

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[1] Cooperative investigation of the Crops Research Division, Agricultural Research Service, United States Department of Agriculture, and the Georgia Agricultural Experiment Stations.

[2] Assistant Nematologist and Nematologist, respectively, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Tifton, Georgia.

[3] DD mixture contains about 50 percent by weight 1,3-dichloropropene and the remainder is mostly 1,2-dichloropropane. Ethylene dibromide (dibromoethane) is available in commercial formulations containing either 41 percent or 83 percent, with naphtha as the diluent. DBCP is 1,2-dibromo-3-chloropropane. It is available commercially in various formulations. DCP is a mixture of technical dichloropropenes. DCP-EDB mixture contains 75.2 percent of 95 percent DCP and 24.8 percent of 83 percent EDB by weight.

of sting nematode can be correlated with the chemical components of nematocides.

MATERIALS AND METHODS

The soil of the field selected for the test was a Tifton sandy loam moderately infested with sting nematodes, Belonolaimus sp. The test was arranged in randomized blocks in which three application rates of five nematocides were replicated four times; unfumigated controls were replicated eight times with two control plots in each block. Each plot consisted of a single row 25 feet in length; the row spacing was 38 inches. Prior to fumigation 4-12-12 fertilizer was applied as a band-application at a rate equivalent to 500 pounds per acre. All nematocides were applied with a hand applicator to assure uniformity of application. Injections were made at a depth of 10 inches and were spaced 12 inches apart along the planting row. Immediately following application of the nematocides, a ridge was formed over each row by use of a disc-hiller plow. The nematocides used, their chemical composition, and weight of active material contained in each gallon of fumigant are listed in Table 1. Rates of application are listed in Table 2.

Henderson Bush lima beans were planted in all plots 2 weeks after soil treatment.

Soil samples were taken from each plot with a soil sampling tube 48, 69, 92, and 126 days after treatment. Each plot sample consisted of 1 quart of soil taken along the row in the root zones of growing lima bean plants. Aliquants of each thoroughly mixed sample were processed by the method of Christie and Perry (4), and the sting nematodes recovered after an extraction period of 48 hours were counted. Yields of lima beans were determined by weighing the beans produced on each plot. Yield data and nematode counts are given in Table 2 as mean values for the various soil treatments.

RESULTS AND DISCUSSION

Plots treated with DCP-EDB mixture, 41 percent EDB or DBCP had significantly lower average populations of sting nematodes during the period of the experiment than did untreated plots, but control differences were not significant between these fumigants at any of the rates used. Populations of sting nematodes in plots treated with all rates of DD mixture and DCP were still low 92 days after treatment, but they were not significantly different from those in untreated plots 126 days after treatment. When similar application rates were compared, control with DCP was not significantly better than with DD mixture, even though DCP contains approximately twice as much dichloropropene per gallon.

At the end of the experiment, populations of sting nematodes were lower in all plots treated with fumigants containing ethylene dibromide than in plots treated with fumigants containing only dichloropropene and dichloropropane. The 9.5 gallons per acre rate of DCP and the 10.9 gallons per acre rate of DCP-EDB mixture contained about the same amount of dichloropropenes, but the latter was more effective for sting nematode control, indicating that the ethylene dibromide fraction was largely responsible for the control difference.

DBCP, which also contains bromine, was as effective in controlling sting nematodes as the materials containing ethylene dibromide.

Although DBCP gave excellent control, yields from DBCP treated plots were low. This was thought to be a stunting effect resulting from applications of higher rates of this nematocide than can be tolerated by lima bean plants.

The different nematocidal responses observed in this experiment may be due either to a more effective initial control by materials containing bromine or to a residual effect of these materials. The data reported herein suggest that DCP-EDB mixture, EDB, and DBCP may have reduced sting nematode populations to a lower level initially than did materials containing dichloropropene; hence a longer period might have been required for the population to become re-established. However, such an interpretation is difficult to verify because present sampling methods limit the evaluation of small population differences that may occur immediately following soil fumigation. A second, more plausible, alternative is that EDB and DBCP remained nematocidally active in the soil longer than materials containing DCP. Support of this view is strengthened when the vapor pressures of these fumigants are considered. DD mixture and DCP have higher vapor pressures than do EDB and DBCP and should, therefore, diffuse from the soil more rapidly, resulting in less exposure of nematodes to the chemicals.

On the basis of the work reported herein the authors conclude that materials containing dichloropropenes were not as effective as materials containing EDB or DBCP in controlling sting nematodes. This test further demonstrates that following soil treatment with dichloro-

Table 1. Chemical composition of soil fumigants tested.[a]

Nematocide	Percentage by weight of chemical components	Pounds per gallon of active material
DD mixture	50 percent 1,3-dichloropropene about 50 percent 1,2-dichloropropane	5.00 about 5.00
DCP	95 percent 1,3-dichloropropene	9.61
DCP-EDB mixture	77.6 percent 1,3-dichloropropene 19.1 percent ethylene dibromide (EDB)	8.46 2.08
41 percent EDB	41 percent ethylene dibromide (EDB)	3.50
DBCP	95 percent 1,2-dibromo-3-chloropropane	17.30

[a]Values were taken or computed from manufacturers' specimen labels.

Table 2. Effect of soil fumigants on control of sting nematodes and on yields of Henderson Bush lima beans.

Soil nematocide	Rate of application			Sting nematodes recovered on indicated sampling day following soil treatment[a]				Bean yields[a]
	Technical material (gallons per acre)	Dichloropropenes (pounds per acre)	Ethylene dibromide (pounds per acre)	48	69	92	126	(ounces per plot)
DD mixture	9.5	47.5	0	1.3	1.8	3.8	58.3	119.3
	12.0	60.0	0	0.3	3.0	3.3	53.0	116.0
	14.5	72.5	0	1.5	0.8	0.8	34.3	127.0
DCP	9.5	92.1	0	0.3	1.8	3.5	37.5	109.5
	12.0	116.3	0	0.3	0.3	1.0	33.5	116.3
	14.5	140.5	0	0.3	1.0	1.0	25.5	105.0
DCP-EDB mixture	6.9	58.4	14.4	0	0	0.5	16.3	147.8
	8.9	75.3	18.5	0.3	0	0	3.8	142.0
	10.9	92.2	22.7	0	0	0	5.5	143.3
41 percent EDB	9.5	0	33.3	0	0	0	1.3	158.0
	12.0	0	42.0	0.3	0	0	2.8	164.0
	14.5	0	50.7	0	0	0.3	3.3	135.5
DBCP	.95	0	0	0.8	0.3	0.3	1.0	117.8
	1.20	0	0	1.2	0.3	1.3	3.8	128.3
	1.45	0	0	0.8	0	0	1.8	104.8
Untreated controls	0	0	0	11.5	15.1	6.3	43.3	76.8
Significance				**	**	**	**	**
LSD (.05 level)				4.2	3.2	3.2	25.9	35.8

[a] Figures given are averages of four replications (plots).

propene materials, sting nematodes can increase in sufficient numbers to reduce yields of lima beans. This calls attention to the possibility that preplant treatment with fumigants that give an extended period of control may in some cases be preferred to treatments with fumigants that give control for only a short period after application.

The question of possible nematocidal specificity of soil fumigants has not yet been fully explored. Later work may show whether apparent residual control by fumigants is due to a comparatively long period of exposure of nematodes to the lethal effect of nematocides, or to prevention of reproduction of nematodes by initial control of one or more stages in the life cycle of these organisms.

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CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, NEMATOLOGY FIELD LABORATORY, TIFTON, GEORGIA

THE CITRUS NEMATODE AND ITS CONTROL ON LIVING CITRUS IN ARIZONA[1]

Harold W. Reynolds and John H. O'Bannon[2]

Abstract

Experiments with an emulsifiable formulation, 1,2 dibromo-3-chloropropane, in irrigation water on living citrus trees were conducted. At all rates tested this chemical gave excellent control of the citrus nematode. "Hedging" and pruning of the trees in conjunction with the soil treatment resulted in rapid tree recovery. Two methods of application were used; the most successful was using a constant head vessel for metering the chemical through a centrifugal pump.

Since 1948 survey work has been conducted in Arizona to determine the extent and damage to citrus by the citrus nematode, Tylenchulus semipenetrans Cobb, 1913. More than half of the groves examined in Arizona have been found to be infected by this nematode. The fact that there are no obvious root symptoms to indicate the presence of this pest on nursery stock -- and therefore it is often not noticed -- may account for its wide distribution. Certain symptoms, however, indicate the presence of this parasite if infected roots are examined closely. Particles of soil adhere to infected roots and make them look larger in diameter at points of infection. Adherence of the soil is due to a gelatinous matrix in which the eggs of the nematode are embedded. Except for lumps of adhering soil particles the infected roots look only slightly enlarged at these places because the surface is rough instead of smooth as on normal roots. The outer portion of infected roots often separates readily from the axial portion at the infected areas.

Advanced above-ground symptoms are yellowing of the leaves and general symptoms of malnutrition (small leaves, small and nonuniform fruit, and defoliated branch ends). Degree of decline varies considerably from grove to grove and within groves. Not all trees with large populations of citrus nematodes on or near their roots show advanced stages of decline. Often the correlation between nematode populations and decline symptoms is negative. Once the parasite is introduced, large populations build up on the healthy, extensive roots of vigorously growing trees, but these populations decrease rapidly as the roots are damaged, become decayed, and decrease in number. Thus trees in an advanced stage of decline may have much smaller populations of citrus nematodes than those with an abundance of healthy roots on which the nematodes can feed. Decline of the above-ground parts may not be noticeable until 3 to 5 years after infection, the period depending upon the care of the grove and the general overall vigor of the trees.

Experiments were conducted in three groves with 1, 2 dibromo-3-chloropropane (DBCP)[3] metered in irrigation water. The objectives of the experiments were 1) to determine optimum application of DBCP for effective citrus nematode control, 2) to determine the effectiveness of this chemical on the citrus nematode when applied to entire rows by metering the chemical into the irrigation water, 3) to test various application methods, 4) to observe response of trees to the chemical and 5) to compare the effects of pruning on the rate of tree recovery in DBCP-treated and untreated trees.

PROCEDURE AND RESULTS

Experiment 1 was set up at the University of Arizona Citrus Experiment Station, Yuma, Arizona in March 1957. The area selected was a block of 36-year-old grapefruit trees on sour orange rootstock moderately to heavily infected with the citrus nematode. The soil of the grove

[1] Cooperative investigations by Crops Research Division, Agricultural Research Service, U. S. Department of Agriculture, and the University of Arizona Agricultural Experiment Station.

[2] Nematologists, Crops Research Division, Tempe, Arizona.

[3] Furnished by the Shell Chemical Corporation. Active ingredient 1,2 dibromo-3-chloropropane containing 2 quarts (8. 6 pounds) active ingredient per gallon in an emulsifiable concentrate.

was Superstition sandy loam covered with a 6- to 10-inch layer of silt deposited by irrigation water.

The DBCP emulsifiable formulation was applied at the rates of 10 and 5 gallons per acre, or 86 and 43 pounds, respectively, of the active ingredient. Applications were made with an engine-driven centrifugal pump that discharged the chemical mixed with irrigation water into individually bordered rows. This area was approximately 23 feet wide and 275 feet long. Twelve trees in a row on 23-foot centers were treated in this manner. The metering of the chemical was timed so that it was applied during the entire period of irrigation. Approximately 5 inches of irrigation water was applied to each plot. Three plots were treated at each of the two rates of application and three control plots received only irrigation water.

One year after the DBCP treatment, trees in treated and untreated plots were randomly selected and pruned. Soil and root samples were collected at intervals from DBCP-treated and untreated plots so that the effectiveness of the treatments on nematode control could be determined. Citrus nematode counts were determined by the Baermann funnel technique.

Citrus nematode counts made prior to and at various intervals after treatment from soil and root samples chosen at random from treated and untreated plots are presented in Table 1.

Table 1. Average citrus nematode populations recovered from 100-gram soil and root samples collected at random in citrus grove before treatment and from DBCP-treated and untreated plots at intervals after treatment.

Treatment	Average nematode population				Percent control after 15 months
	Before treatment	After treatment			
		5 months	9 months	15 months	
10 gallons	4034	0	1	1	99.9
5 gallons	4034	0	0	3	99.9
Control (water only)	4034	5322	9800	5900	0

Moderately high citrus nematode populations were present in all plots before treatment. Both rates of application gave excellent control 5, 9 and 15 months after treatment. Effective control was obtained over the entire row regardless of the distance down the row from the point where the chemical was introduced into the water. Untreated plots continued to show moderately high populations. There was no visible evidence of tree recovery of the DBCP-treated over the untreated trees at the end of the first growing season, nor was any effect due to pruning noted at 4 months after this operation.

Experiment 2 was established in November 1957 in cooperation with Mr. G. C. Morse, a citrus grower at Yuma, Arizona. A portion of a 10-acre citrus grove was selected. The trees were 31-year-old grapefruit on sour orange rootstock moderately to heavily infected with the citrus nematode. The soil was a Superstition sandy loam covered with a 6- to 10-inch layer of silty loam. Approximately 3 acres were divided into plots of three rows each. Each row consisted of 15 trees on 22-foot centers. DBCP formulation was applied at the rates of 6 and 3 gallons per acre containing 51.6 and 25.8 pounds, respectively, of the active ingredient. The treatments were made in irrigation water applied at the head of the rows with three rows at a time receiving treatment. Each row was bordered laterally as described for experiment 1.

A 200-gallon Bean sprayer filled with water and emulsifiable DBCP was used for treatment. The DBCP-water emulsion was constantly agitated during the application. Irrigation water was diverted from the irrigation valves into the three rows to be treated. Three hoses were extended from the spray tank to the rows receiving treatment. The emulsified material was metered into the irrigation water and the metering was timed to extend over the entire period of irrigation; approximately 5 inches of water was applied during the irrigation period. Two plots each were treated at each of the two rates of application, and two control plots received irrigation water only.

Approximately 3 months after treatment, all trees in the treated and untreated plots were "hedged." Power-driven saws on a vertical boom were used in the hedging operation. The saws were pulled up and down and across the rows so that four sides of the trees were cut back; branch ends up to 18 inches in length were removed. Selective pruning and topping with hand-pruning saws followed this operation.

Soil and root samples were collected from treated and untreated plots and the effectiveness of the treatments on nematode control was determined by the same method used in experiment 1.

Citrus nematode populations as determined from soil and root samples taken 8 months after treatment showed excellent control of the citrus nematode in all DBCP treated plots (Table 2).

Table 2. Average citrus nematode populations recovered from 100-gram soil and root samples collected at random in citrus grove before treatment and from DBCP-treated and untreated plots 8 months after treatment.

Treatment	Average nematode population		Percent control
	Before treatment	8 months after treatment	
6 gallons	6283	6	99.7
3 gallons	6283	1	99.9
Control	6283	2200	0

Evidence of tree recovery was definitely observed in the treated plots as early as 7 months after treatment and 4 months after hedging and pruning. New and vigorous flushes of growth were evident throughout the treated plots, but only slight recovery and very little new growth were observed in untreated plots.

Experiment 3 was established in April 1958 in cooperation with Mr. W. A. Nelson, Manager of the Hearsh Brothers citrus groves at Yuma, Arizona, in part of a 10-acre citrus grove. The trees were 30-year-old grapefruit on sour orange rootstock heavily infected with the citrus nematode. The soil was very similar to that described for experiments 1 and 2. Approximately 2 1/2 acres was divided into plots of four rows each, consisting of 15 trees per row on 22-foot centers. The DBCP formulation was applied at the rates of 6, 4 and 2 gallons per acre, or 51.6, 34.4 and 17.3 pounds, respectively, of the active ingredient. Each row was bordered laterally as described in experiment 1. An engine-driven centrifugal pump of 4000 gallons per hour capacity was used in application. The emulsifiable DBCP was metered through a drilled orifice from a constant head vessel into the inlet side of the centrifugal pump as described in experiment 1. A small portion of water was diverted from a concrete lined irrigation ditch through the pump and the DBCP water emulsion was discharged back into the irrigation ditch. The irrigation water was then diverted into the bordered citrus rows receiving treatment. Four rows were treated at a time. Metering of the DBCP water emulsion was timed to extend over the entire period of irrigation, with approximately a 5-inch irrigation applied. There were three treated plots and one control plot that received irrigation only. No hedging or hand pruning was done.

Soil and root samples collected from DBCP-treated and untreated plots show essentially complete citrus nematode control by the 4-and 6-gallon per acre rates (Table 3). At the 2-gal-

Table 3. Average citrus nematode population recovered from 100-gram soil and root samples collected at random in citrus grove before treatment and from DBCP-treated and untreated plots 2 months after treatment.

Treatment	Average nematode population		Percent control
	Before treatment	2 months after treatment	
6 gallons	10,000	0	100.0
4 gallons	10,000	1	99.9
2 gallons	10,000	60	99.4
Control (water only)	10,000	10,000	0

lon rate a very slight citrus nematode population was recovered. No visible tree recovery was observed 3 months after treatment.

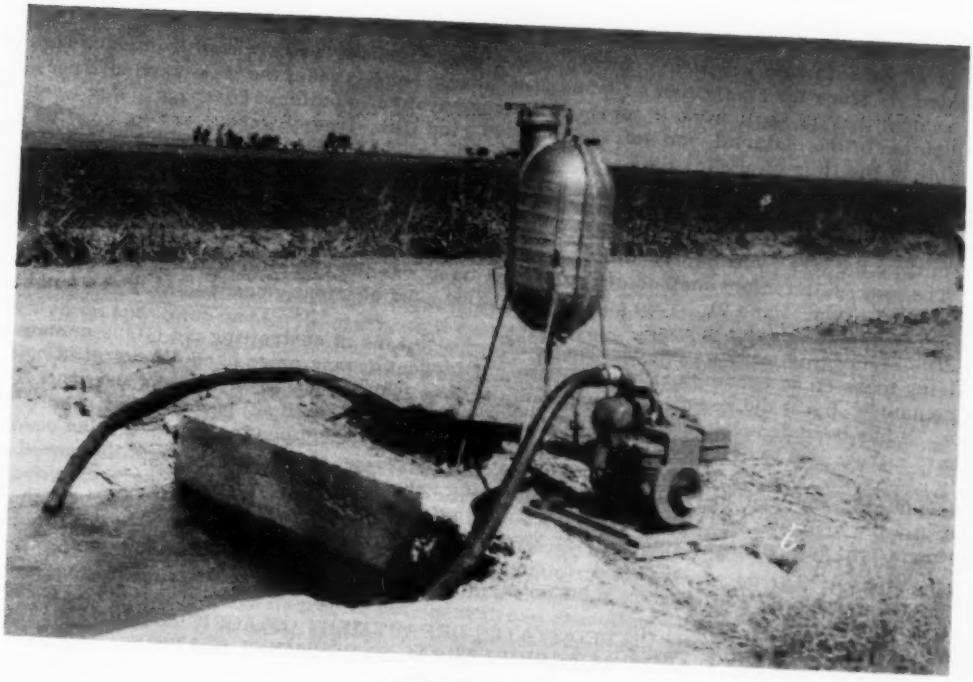

FIGURE 1.

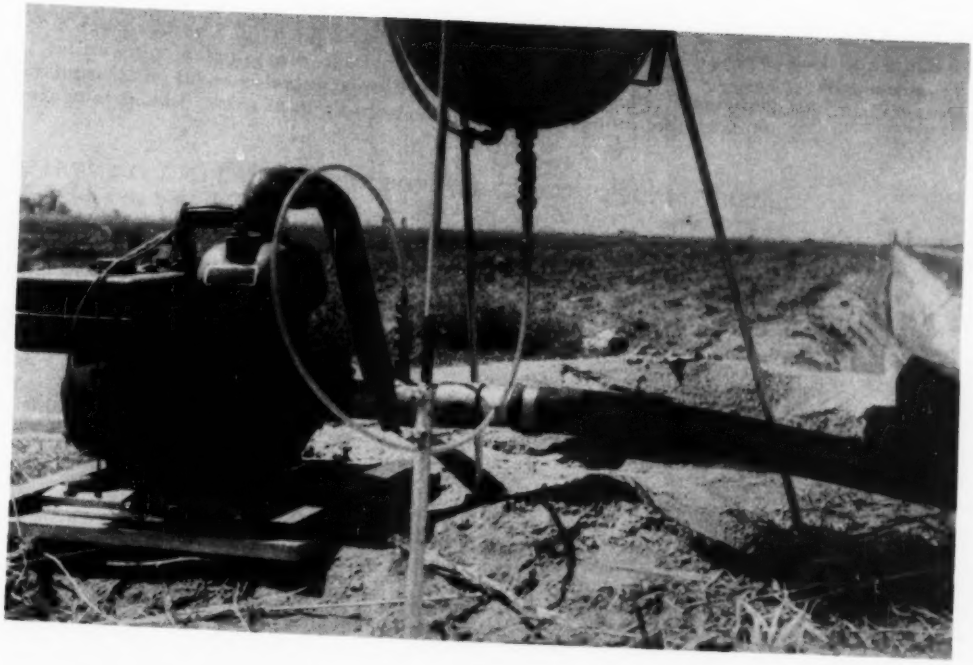

FIGURE 2.

DISCUSSION

In all experiments excellent control of the citrus nematode was secured from each rate of application. Since the emulsifiable formulation flowed readily under the "low-skirted" trees, it was evident that its application in irrigation water resulted in uniform coverage. For adequate distribution of the material, the soil should be in good tilth, free of weeds, and as level as possible at the time of application.

Hedging or selective pruning of trees or both combined with DBCP treatment aided in rejuvenating debilitated trees and resulted in a more rapid tree recovery. It has been observed that pruning alone without citrus nematode control may result in temporary tree recovery, but that in time the trees will revert into a status of decline.

The most satisfactory method of application tried was the centrifugal pump and constant head vessel (Figs. 1 and 2). This equipment can be used efficiently regardless of the number of rows irrigated at any one time.

The aim of this work was to develop an effective means of controlling the citrus nematode on living trees on an economical basis. Even the largest dosage rates used did not eliminate the nematode, but it did reduce the populations to less than 1 percent on those of the control plots. It has been demonstrated that DBCP at higher rates of application than those used is toxic to citrus. No toxicity symptoms were noted in these experiments. However, the possibility of toxicity cannot be eliminated. This factor, as well as economy, suggests the use of moderate or lower, rather than higher, application rates. Failure to kill all the nematodes, even with the highest rates used, suggests that in practice it may be necessary to repeat the treatment at lighter dosage rates in later years.

CROPS RESEARCH DIVISION, UNITED STATES DEPARTMENT OF AGRICULTURE, AND THE UNIVERSITY OF ARIZONA AGRICULTURAL EXPERIMENT STATION, TEMPE

LONGEVITY OF XANTHOMONAS MALVACEARUM IN DRIED COTTON PLANTS

Peter A. Ark[1]

Abstract

Xanthomonas malvacearum was recovered in culture from lesions of black arm disease on dried cotton plants stored at room temperature for 6 years.

Xanthomonas malvacearum, the cause of the black arm disease of cotton, is known to be carried on and in the seed. However, to the writer's knowledge, information on its survival in the plant is not to be found in plant pathological literature. It seemed that such information might be of interest to phytopathologists concerned with this crop.

In September 1952, typical cultures of X. malvacearum were isolated from two large cotton plants having numerous lesions on the leaves, bolls, and other plant parts. These cultures proved to be pathogenic to young greenhouse-grown cotton plants when they were inoculated by spraying with heavy bacterial suspensions and held under suitable temperature and moisture conditions.

The plants from which the original isolations were made were placed in paper grocery bags and stored at room temperature (24° to 26°C) for future reference. Recently a few black lesions on these dried specimens were cultured in potato-dextrose-peptone agar by the serial dilution method. Numerous yellow-colored bacterial colonies appeared on all plates after 4 days. Several colonies were transferred to potato-dextrose agar slants and incubated at 28°C for 2 days. Abundant shiny growth, typical of X. malvacearum, was produced. A heavy suspension of the organism, in distilled water, was sprayed on young greenhouse-grown cotton plants which subsequently were held for 24 hours in a moist chamber under about 90 percent relative humidity. At the end of this incubation period the inoculated plants were set on the bench in a greenhouse with a relative humidity of 75 to 80 percent and temperature fluctuating between 70° and 90°F. Small water-soaked lesions appeared on both the cotyledons and the small true leaves after 3 days. After 7 to 10 days the lesions were well defined and, when cultured, yielded typical yellow colonies on potato-dextrose-peptone agar plates after about 4 days incubation at 28°C. Upon inoculation these colonies proved to be X. malvacearum. Therefore, X. malvacearum survived at least 6 years in dry cotton plants stored at room temperature.

DEPARTMENT OF PLANT PATHOLOGY, UNIVERSITY OF CALIFORNIA,
BERKELEY 4, CALIFORNIA

[1] Professor of Plant Pathology, University of California, Berkeley 4, California.

THE INCIDENCE OF ANTHRACNOSE FUNGI ON ORNAMENTAL FOLIAGE PLANTS IN WASHINGTON STATE GREENHOUSES

Shirl O. Graham and James W. Strobel[1]

Abstract

Surveys in 1956, 1957 and 1958 have disclosed an increased incidence of anthracnose fungi on ornamental foliage plants in Washington State greenhouses. Gloeosporium, Colletotrichum and Glomerella have been identified and isolated from twenty hosts. Certain of the hosts harbored two or three of these anthracnose genera, while other hosts were attacked by only one genus. The hosts and the anthracnose genera found are summarized. New United States or Washington State records are indicated.

Owing to increased public demand for more and newer varieties of ornamental foliage plants, and a shift from cut flower production to pot plant sales in the Pacific Northwest, several wholesale enterprises in this industry have been created in the State of Washington. Growing with the industry have been erratic outbreaks of leafspot diseases. Surveys made in 1956 and repeated in both 1957 and 1958 showed that, almost exclusively, the leafspots were caused by anthracnose fungi.

In 1956 spotting occurred on but two hosts, Syngonium podophyllum and Ficus elastica var. variegata. Identifications and isolations were solely Gloeosporium spp. Several surveys made during 1957 disclosed anthracnose fungi attacking 11 additional hosts that had not evidenced the disease before. Identifications placed the genera of these pathogens in four combinations. Seven hosts (Cryptanthus bromelioides, Cryptanthus bromelioides var. tricolor, Ficus elastica, Ficus elastica var. variegata, Philodendron cordatum, Philodendron hastatum, and Philodendron panduraeforme) were attacked by Gloeosporium. Three hosts (Aucuba japonica var. crotonifolia, Aglaonema commutatum, and Dracaena godseffiana) were attacked by Colletotrichum. Two hosts (Dracaena fragrans var. massangeana and Syngonium podophyllum) were attacked by Gloeosporium and Colletotrichum. One host (Dieffenbachia amoena) was attacked by Gloeosporium, Colletotrichum and Glomerella all on the same infected plant.

In 1958 seven additional hosts were found harboring anthracnose fungi. The same erratic combinations occurred. One host (Stephanotis floribunda) was attacked by Gloeosporium. Two hosts (Dracaena sanderiana and Monstera punctulata) were attacked by Colletotrichum. Three hosts (Dieffenbachia picta, Philodendron pertusum (Monstera deliciosa), and Peperomia obtusifolia) were attacked by Gloeosporium and Colletotrichum, and one host (Ficus pandurata) was attacked by a different combination, Gloeosporium and Glomerella.

The presence of Glomerella cingulata, the perfect stage of the Colletotrichum-Gloeosporium "complex" on Dieffenbachia, introduces the complicating factor of hybridization. New strains with increased host ranges can develop, because Dieffenbachia is grown in close quarters with most other foliage plants, and usually under intense culture as well.

A summary of the hosts and the anthracnose genera found on them is given in Table 1. All the reports listed in the table are new records either for the State of Washington or for the United States. Host determinations were based on Graf (2) and Bailey (1).

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[1] Assistant Plant Pathologist and Research Assistant, respectively, State College of Washington, Pullman.

Work conducted under project No. 510, Washington Agricultural Experiment Stations.

Table 1. Summary of anthracnose fungi found to be attacking ornamental foliage plants in Washington State greenhouses, including new host records[a].

Host	Gloeosporium	Colletotrichum	Glomerella
Aucuba japonica var. crotonifolia		W	
Aglaonema commutatum		US	
Cryptanthus bromelioides	US		
Cryptanthus bromelioides var. tricolor	US		
Dieffenbachia amoena	W	W	W
Dieffenbachia picta var. Rudolph Roehrs	W	W	
Dracaena godseffiana		W	
Dracaena fragrans var. massangeana	W	W	
Dracaena sanderiana		W	
Ficus elastica	W		
Ficus elastica var. variegata	W		
Ficus pandurata	W		W
Monstera punctulata		W	
Peperomia obtusifolia	US	US	
Philodendron cordatum	US		
Philodendron hastatum	US		
Philodendron panduraeforme	US		
Philodendron pertusum (Monstera deliciosa)	US	US	
Stephanotis floribunda	US		
Syngonium podophyllum	US	US	

[a] US = New record for United States; W = New record for State of Washington

Although Weiss (4) reports Glomerella cincta on Dieffenbachia picta, it is not certain if this report includes imperfect stages of the organism as well as the ascigerous stage. We have identified Colletotrichum, Gloeosporium and Glomerella on our collections of Dieffenbachia amoena. On Dieffenbachia picta var. Rudolph Roehrs we have observed the imperfect stages, Gloeosporium and Colletotrichum. These reports are listed as new for Washington. Seymour (3) reports Glomerella cincta on Dracaena terminalis and Colletotrichum dracaenae on Dracaena sp. indet. Weiss (4) has listed Colletotrichum, Gloeosporium and Glomerella as occurring on Dracaena spp. Our reports of Colletotrichum on Dracaena godseffiana, Dracaena sanderiana, and Dracaena fragrans var. massangeana thus must be considered new reports for Washington, along with our report of Gloeosporium on Dracaena fragrans var. massangeana.

Glomerella cincta and Colletotrichum cingulatum are reported on Ficus elastica by Seymour (3). As synonomy under Colletotrichum cingulatum he lists two species of Gloeosporium and Glomerella cingulata. Weiss (4) lists only Glomerella cingulata on Ficus elastica and Ficus elastica var. variegata. Our reports of Gloeosporium on Ficus elastica, Ficus elastica var. variegata and both Gloeosporium and Glomerella on Ficus pandurata are considered new State records.

Gloeosporium has not been previously reported on the various Philodendron species in this country although Weiss (4) has reported Colletotrichum on Philodendron spp. Thus, our reports of Gloeosporium on Philodendron cordatum, Philodendron pertusum (Monstera deliciosa), Philodendron hastatum and Philodendron panduraeforme are apparently new for this country. New State records of Colletotrichum were identified on Monstera punctulata and Philodendron pertusum (Monstera deliciosa).

On six hosts our reports of anthracnose are new for both the host genus and species. As indicated in Table 1. these are considered new United States reports. Colletotrichum and Gloeosporium were identified on Syngonium podophyllum and Peperomia obtusifolia. Gloeosporium was found attacking Stephanotis floribunda, Cryptanthus bromelioides, and Cryptanthus bromelioides tricolor. On the sixth host, Aglaonema commutatum, Colletotrichum was identified.

Our collection of Colletotrichum on Aucuba japonica var. crotonifolia is reported as a new Washington State record, because Weiss (4) has reported Colletotrichum on this host from other areas of the country.

The variable identifications listed in the table are not surprising; observations of this nature have been made on many other host genera too numerous to cite. The identifications stress the

innate variability of the imperfect stages of Glomerella and introduce a question on the tenability of using setae production as a stable, and hence valid, morphologic character to separate Colletotrichum from Gloeosporium.

The variability of the imperfect stages, along with the increasing host range, prompted the initiation of experiments on the host range, cultural characteristics and setae production of the isolates. Preliminary results of these studies are forthcoming.

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WASHINGTON AGRICULTURAL EXPERIMENT STATIONS, PULLMAN

NEW FUNGICIDES FOR THE CONTROL OF BLACKSPOT ON ROSES

T. J. Henneberry and R. V. Travis[1]

Since 1950 studies have been conducted at Beltsville, Md., to develop combination sprays and dusts for the control of pests on outdoor roses. Several effective miticide and fungicide combinations have been developed and recommended for use (1, 2, 3). This paper is the report of an experiment conducted in 1957 to evaluate several new fungicides for the control of blackspot, caused by Diplocarpon rosae Wolf, when used in combination sprays with effective miticides and insecticides.

METHODS

The experiment was conducted in a randomized block design consisting of four blocks of 11 plots each. One plot consisted of six plants, three each of the varieties Helen Traubel and Red Radiance. The plants were 2 1/2 feet apart with 5 feet between plots. The plants were mulched with 1 to 2 inches of sawdust and maintained in good growing condition with fertilizer and periodic irrigations throughout the season.

The treatments were a factorial arrangement of the fungicides Phaltan (n-trichloromethylthiophthalimide), Cyprex (n-dodecylguanidine acetate), Agri-mycin 500 (streptomycin 67 ppm, copper 800 ppm, oxytetracycline 6.7 ppm) and zineb (zinc ethylene bisdithiocarbamate) in combination with the miticides Aramite and malathion. Pfizer P-189 and Anisomycin were used in combination sprays with Aramite.

The sprays consisted of the various combinations of miticide and fungicide each at the strength of 2 pounds of wettable powder in 100 gallons of spray as follows: 15 percent Aramite, 25 percent malathion, 65 percent zineb, 70 percent Cyprex, 50 percent Phaltan, and Agri-mycin 500. Solutions of P-189 and Anisomycin were added to the sprays to give a concentration of 50 ppm. All sprays including the check spray also contained 1 pound of DDT, 1/2 pound of lindane, and 1/2 pint of the wetting agent Santomerse per 100 gallons. They were applied with a 2-gallon compressed-air sprayer at 60 pounds per square inch.

The sprays were applied first on July 17 and weekly thereafter until September 17. Plants in plots were severely infected with blackspot when the treatments were started.

Blackspot ratings were made August 29 after the seventh application, and on October 9, 3 weeks after the tenth and last application.

RESULTS

The results are presented in Table 1.

On August 29 Phaltan, Cyprex, and zineb had reduced blackspot 42 percent to 60 percent below the check. P-189, Anisomycin, and Agri-mycin 500 were ineffective. On October 9, Phaltan and Cyprex had reduced blackspot infections more than 65 percent below the check. Both these materials were significantly more effective than zineb, apparently because of a longer residual action. P-189, Anisomycin, and Agri-mycin 500 were still ineffective.

Phaltan and Cyprex were compatible with Aramite and malathion. No spray injury or reduction in miticidal activity was observed. However, both fungicides left conspicuous white residues on the foliage, which may be objectionable on ornamentals such as roses.

[1]Respectively, Entomology Research Division, and Crops Research Division, Agricultural Research Service, United States Department of Agriculture.

Table 1. Percent of foliage infected with blackspot on rose plots treated with a miticide and fungicide combination spray.

Treatment	August 29	October 9
Aramite plus -		
Phaltan	53	29
Cyprex	53	30
Agri-mycin 500	88	80
Zineb	53	50
Pfizer P-189	93	98
Anisomycin	98	98
Malathion plus -		
Phaltan	45	30
Cyprex	40	33
Agri-mycin 500	88	83
Zineb	58	57
Check	100	100
LSD 5 percent	17	11

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ENTOMOLOGY RESEARCH AND CROPS RESEARCH DIVISIONS, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE

RECORDED DUTCH ELM DISEASE DISTRIBUTION IN NORTH AMERICA AS OF 1957[1]

Francis W. Holmes[2]

Additional discoveries of Dutch elm disease have made obsolete last year's revision (4) of the distribution map initially published in 1956 (3). During 1957, the disease was found for the first time in Iowa (5), Kansas (6), and New Brunswick (1).

Publication of a third version of the map to show these findings (Figure 1) affords an opportunity to include many additional locations in other areas, especially in Illinois, Michigan, West Virginia, Virginia, Delaware, Vermont, New Hampshire, and Maine.

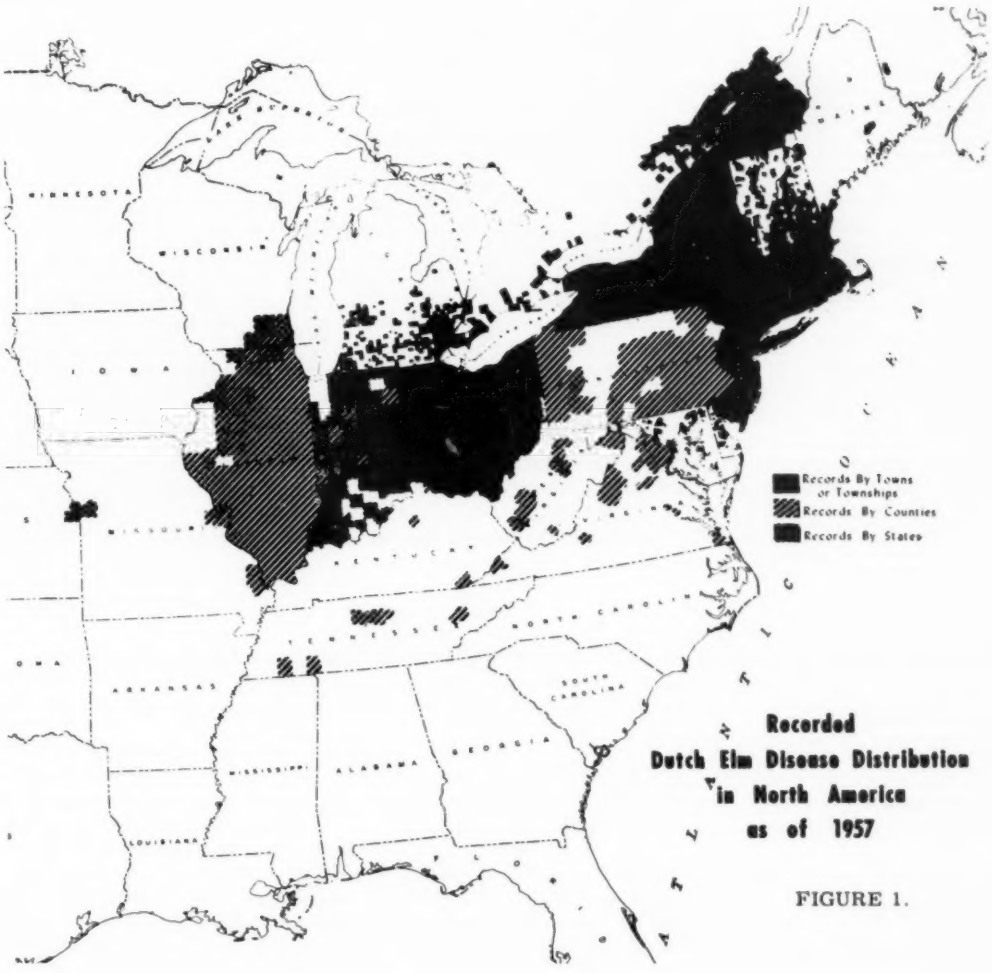
Records By Towns or Townships
Records By Counties
Records By States
Recorded Dutch Elm Disease Distribution in North America as of 1957

FIGURE 1.

[1] Contribution No. 1169 of the University of Massachusetts, College of Agriculture, Experiment Station, Amherst, Massachusetts.

[2] Assistant Professor, Shade Tree Laboratories, Department of Entomology and Plant Pathology, University of Massachusetts.

The format for Indiana has been changed to indicate that knowledge of Dutch elm disease distribution in most of that State is based on symptom observation only, but that culture records exist for locations in five Indiana counties. The format for Connecticut has been changed to indicate the existence of published culture records for most Connecticut towns (2).

This map is intended to serve not merely as an approximate indication of disease distribution, but also as an accurate record of localities of diseased elms from which culture isolations of the fungus Ceratocystis ulmi (Buisman) Moreau have been made. Wherever possible, therefore, data have been plotted on a town basis. Where town records were not available, county records were used. Only as a last resort were larger areas treated as single units.

Nevertheless, it should be pointed out that this map does not purport to show accurately where trees died of the Dutch elm disease in any one year. In many of the locations where this disease has been found in past years, no cases may have occurred recently. A notable example of this situation is Colorado, where the Dutch elm disease was found in Denver in 1948. Although the European elm bark beetle is still present, the disease has not been found recently. (To allow the use of a larger scale, Colorado is omitted from the present map.) On the other hand, it is likely that the Dutch elm disease occurs in many unmarked localities, especially near the borders of or within the regions where it has been recorded frequently.

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SHADE TREE LABORATORIES, UNIVERSITY OF MASSACHUSETTS, AMHERST

NEW OR UNUSUAL RECORDS OF PLANT DISEASES

TOBACCO DISEASES IN PANAMA

By G. B. Lucas[1]

In 1958 flue-cured, Burley and criollo (native) tobacco were being grown in Panama, principally in Chiriqui province. Little, if any, of this tobacco was exported, most of it being used for domestic purposes.

The writer spent a week in November 1957 and a week in January 1958 making a survey of tobacco diseases for Tabacalera Istmena, Panama, R. de P. There appears to be no previous record of occurrence in Panama of some of the diseases encountered. Therefore, the diseases seen in Chiriqui Province, near the town of David, are herein reported.

Nematode Diseases -- Root-knot (Meloidogyne incognita); brown root rot (Pratylenchus sp.). Soil samples from tobacco fields also contained Helicotylenchus sp. and Trichodorus sp[2].

Fungus Diseases -- Damping off (Pythium sp.); frogeye (Cercospora sp.); brown spot (Alternaria longipes); southern stem rot (Sclerotium rolfsii).

Bacterial Diseases--Hollow stalk (Erwinia aroideae); Granville wilt (Pseudomonas solanacearum); frenching (Bacillus cereus).

Virus Diseases -- Mosaic, leaf curl. Other virus diseases not positively identified include etch and veinbanding.

Miscellaneous Diseases -- Weather fleck or "physiological leaf spot"; false broom rape.

Damping-off is a serious plant-bed disease. The use of infected transplants frequently results in poor stands in the field and makes replanting necessary. Root knot and brown root rot, which were encountered frequently, appear to be widespread, while Granville wilt and frogeye also cause much damage; these four diseases are the most important. The most prevalent virus disease was leaf curl. Virus diseases are a constant threat to tobacco production because of the high insect populations, the 12-month growing season, and wild host plants that serve as inoculum sources.

The other diseases appeared to be of little economic importance.

NORTH CAROLINA STATE COLLEGE, RALEIGH

[1] Associate Professor of Plant Pathology, Plant Pathology Department, North Carolina State College, Raleigh.

[2] Dr. J. N. Sasser kindly identified the nematodes.

STINKING SMUT IN VIRGINIA

By S. B. Fenne

District Agent G. H. Clark reported on September 8: "During the past few weeks several millers and wheat buyers have called to my attention the fact that a number of crops this year were affected by stinking smut or bunt. Several crops had to be condemned because of this disease."

After the First World War, stinking smut was perhaps the most serious plant disease in this State, causing several million dollars loss. One of the reasons for the destructiveness of this disease is that even though there may be only a small percentage of stinking smut in a wheat crop, the resulting grain is unsuitable for flour or feeding because of the putrid odor that is easily mixed throughout the whole lot.

Stinking smut was almost eliminated from Virginia in the 1920's because most farmers treated their wheat with chemicals. In those days copper carbonate was used. Now there are many other chemicals that are superior to copper carbonate. The mercurials are best, because not only will they control stinking smut very effectively, but also several other diseases that are common to wheat and other cereals.

Since much of the wheat seed in Northern Virginia is home-grown, it is extremely important that farmers treat their seed before planting with a recommended chemical material. Certified seed or seed purchased from commercial seedsmen usually has been treated. Treated seed should not be retreated. The chances are that if farmers do not treat their seed for the control of stinking smut in one year, in the next year the amount of the disease will increase greatly and will result in serious losses. Stinking smut can be spread easily from farm to farm by threshing machines, combines, and so forth. Wheat seed should first be thoroughly cleaned by fanning to remove smut balls, shrivelled grains, and debris; then it should be chem-

ically treated at the recommended rate for the material used. Treatment should be made at least 24 hours before planting, so that the chemical vapors may penetrate the lot of seed.
EXTENSION SERVICE, VIRGINIA POLYTECHNIC INSTITUTE, BLACKSBURG, VIRGINIA

ANNOUNCEMENT
GOLDEN JUBILEE VOLUME

The 50th Anniversary Committee of the American Phytopathological Society wishes to announce to those who have made prepublication orders for the Volume that a change in the publisher has been made that will result in a slight delay of the publication date. The book will be published by the University of Wisconsin Press. The manuscript is now in their hands and the Volume should be available in the early spring of 1959.

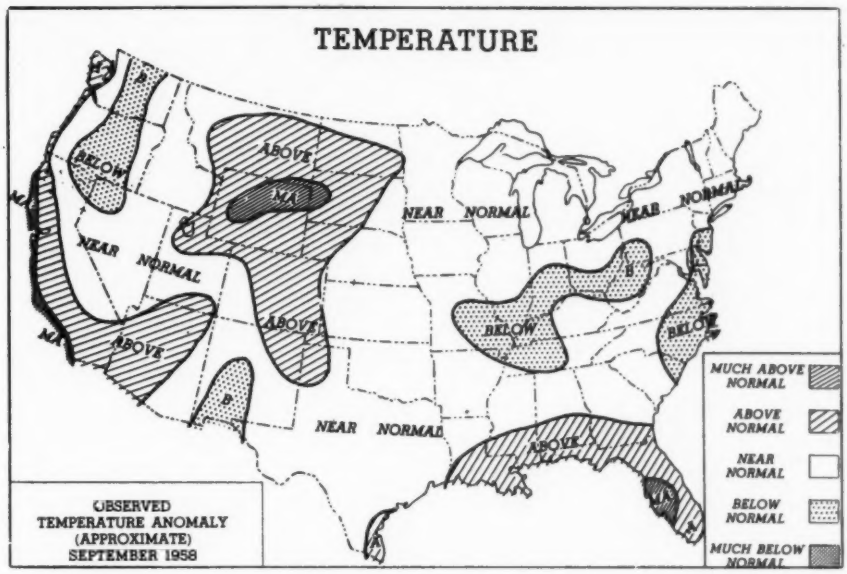
CORRECTIONS

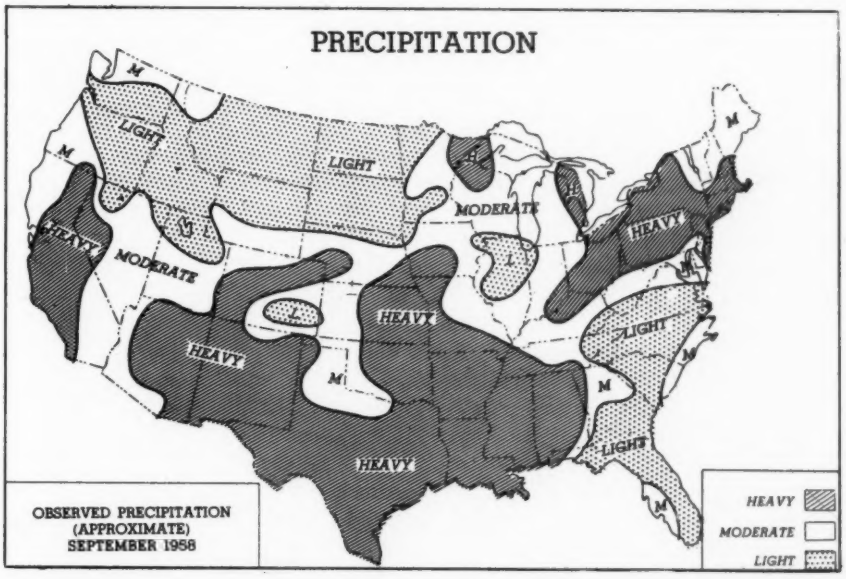
REPORTER, July issue (Volume 42, Number 7), "Physiologic races of Puccinia graminis in the United States in 1957" by D. M. Stewart, R. U. Cotter, and B. J. Roberts, for 11 to 32 race group read 11-32 race group, on p. 881, line 4, and p. 884, line 3. Similarly, for 17 to 29 race group read 17-29 race group, on p. 881, line 5, p. 882, lines 3, 5, and 8, and p. 884, line 8.

REPORTER, August issue (Volume 42, Number 8), page 924: Reference 33 should read, "Roane, C. W., T. M. Starling, and H. H. McKinney."

REPORTER, August issue (Volume 42, Number 8), page 939: Delete in Honduras from the title of Table 1. The table is obviously a summary report. The Reporter acknowledges responsibility for the error.

REPORTER, September issue (Volume 42, Number 9), page 1002: In Figure 3 the abscissa should continue to the lower right corner with the same time interval as in Figures 1 and 2.





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.